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Folia rheumatologica

heumatology in every day practice

Osteoarthritis of the fingers

F. Wollheim

Osteoarthrosis of hip and knee

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The aim of "Folia rheumatologica" is to make a further contribution to the continuing education of doctors in the area of practice-oriented rheumatology.

Concrete case material is used to illustrate the relevant, fundamental medical facts.

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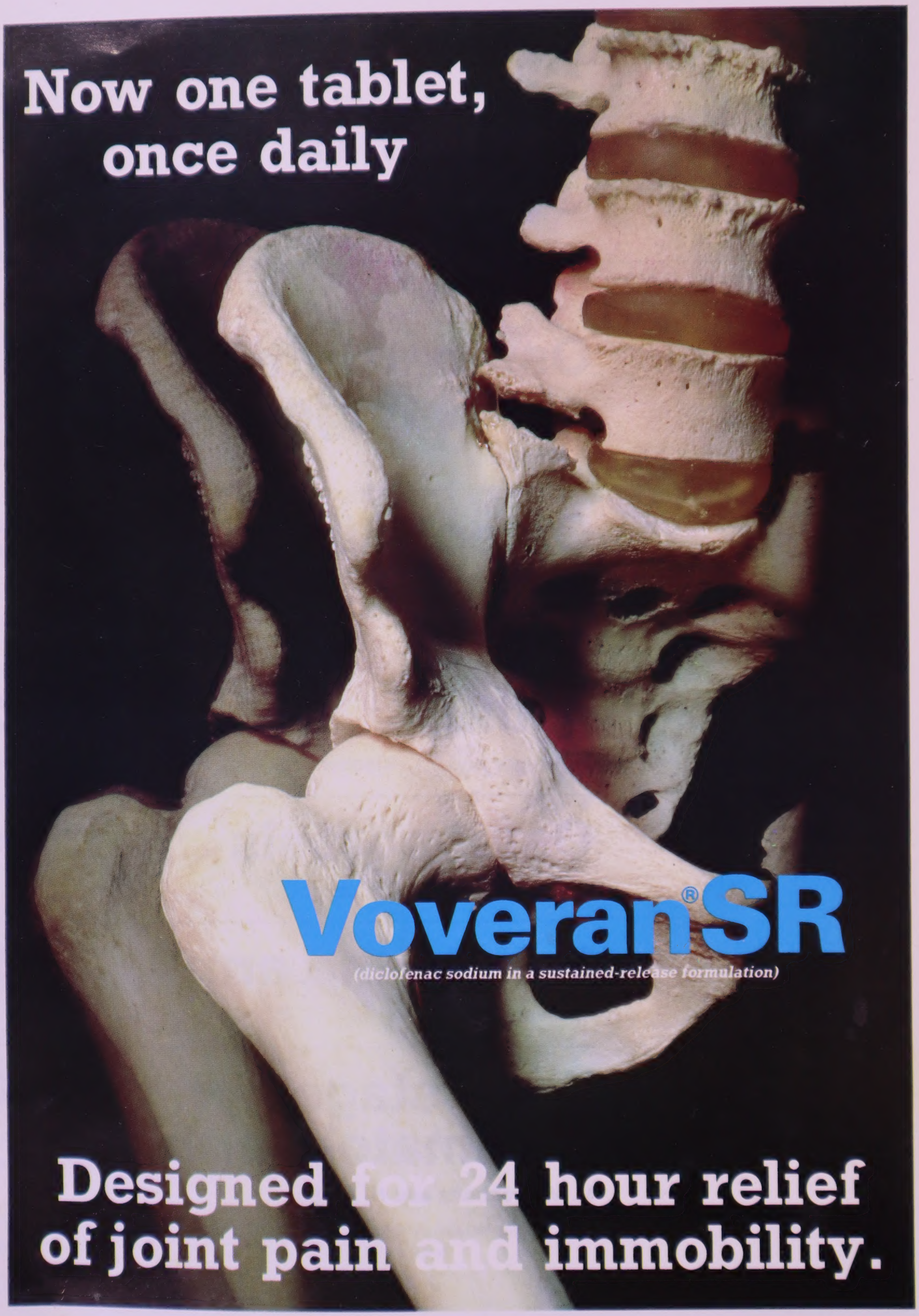
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Osteoarthritis of the fingers

F. Wollheim

Vol.1

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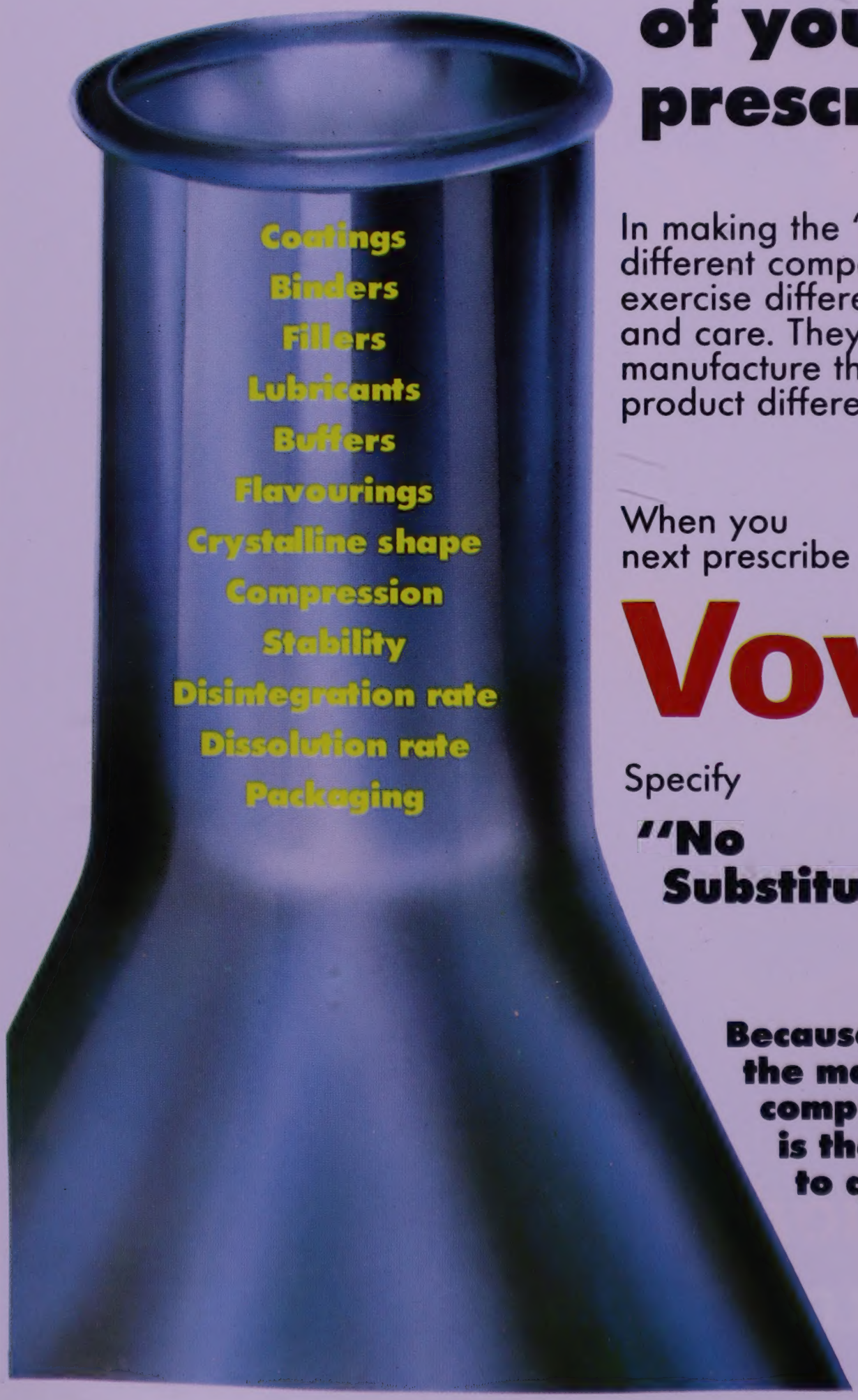

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Osteoarthritis of the fingers

F. Wollheim

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Case 1

Mrs AB, a 54-year-old charwoman

"Doctor, what's wrong with my fingers? I have this pain that's been coming and going for the last 3 or 4 years. And now I have these tender swellings."

"Do you have other joint problems as well?"

"Oh, yes. I have some pain in both shoulders, and in the last few weeks the left elbow has been hurting at work. And then there's this problem with my back."

"Have you had previous checks with a doctor for these complaints?"

"Yes, I've received a couple of cortisone injections, and they've been trying to have me take these pills, of what I understand is enteric coated aspirin."

"Has it helped?"

"Some of the injections were quite effective. The pills didn't do much good, and my stomach doesn't like them."

Examination

Blood pressure was 210/110 mm Hg. Lasègue's test 10° on the right side. Tenderness was found over the left acromioclavicular joint, the lateral epicondyle of the left elbow, the wrists, and two proximal and most distal interphalangeal joints. Flexion of the right middle and the left ring fingers was impaired. Heberden's nodes were seen and palpated on the fifth right finger and on the second to fourth fingers of the left hand.

Laboratory investigations

The erythrocyte sedimentation rate was 12 mm/h. The haemoglobin was 144 g/L. The Waaler-Rose test gave a titre of 1/32, a borderline result. Radiological examination of the back, shoulders, and elbows did not yield any abnormal findings, whereas the hands showed what the radiologist interpreted to be "severe erosive changes of several distal interphalangeal joints".

X-Ray examination

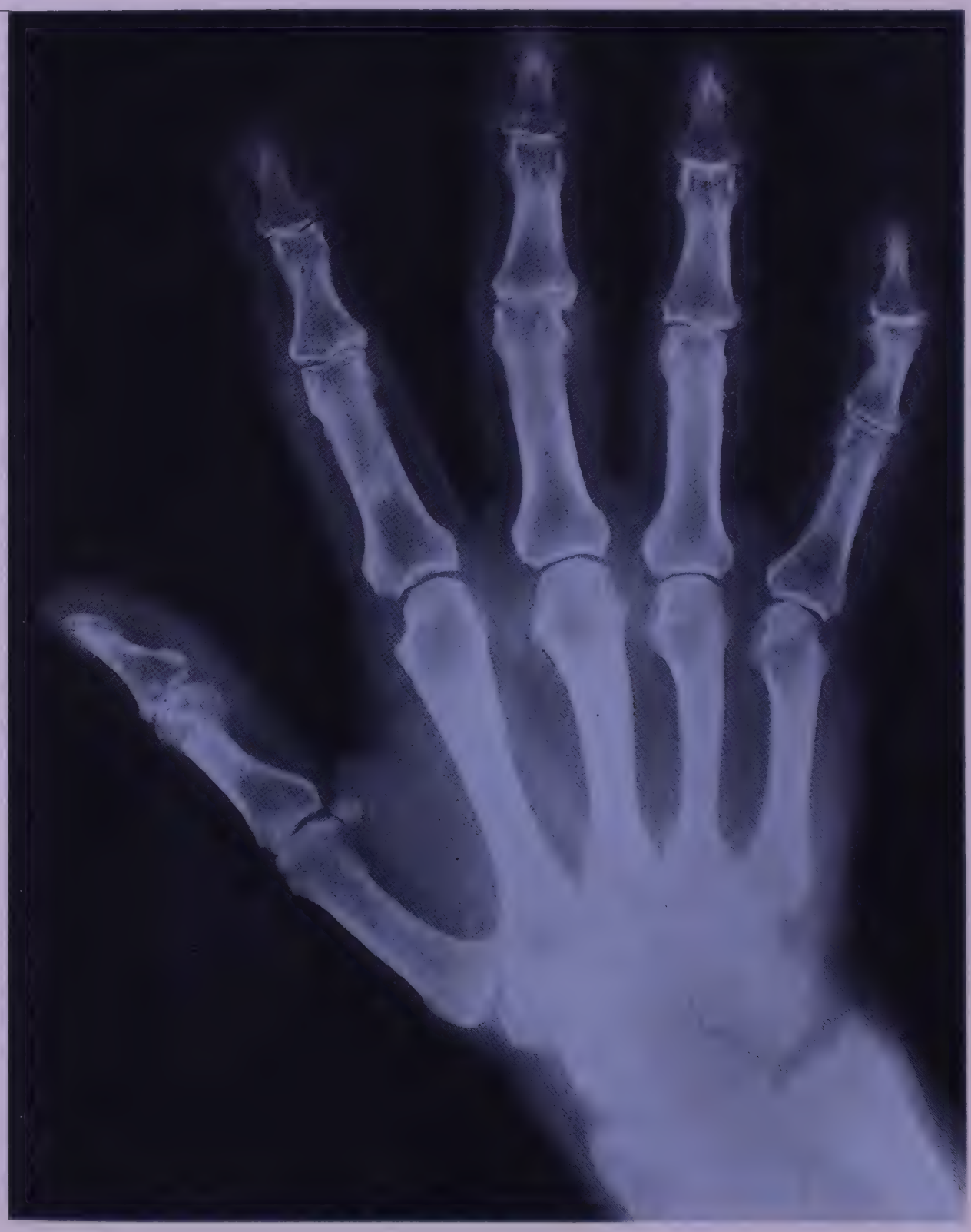
Cartilage reduction of all DIP joints

Osteophytes

Absence of osteoporosis

Intact metacarpophalangeal joints

Right hand radiogram



Synopsis

In spite of the misleading radiologist's report and the borderline Waaler-Rose titre, the correct diagnosis of osteoarthritis of the fingers was made. There were, however, some confusing features of an inflammatory nature in the affected finger joints, as well as pain and tenderness in the elbow and shoulder joints.

Therapy

An adjustment to lighter work and further attempts to find a suitable analgesic drug were made at this stage. The patient was told that she was *not* suffering from rheumatoid arthritis.

Differential diagnosis

Other possible diagnoses that might be considered:

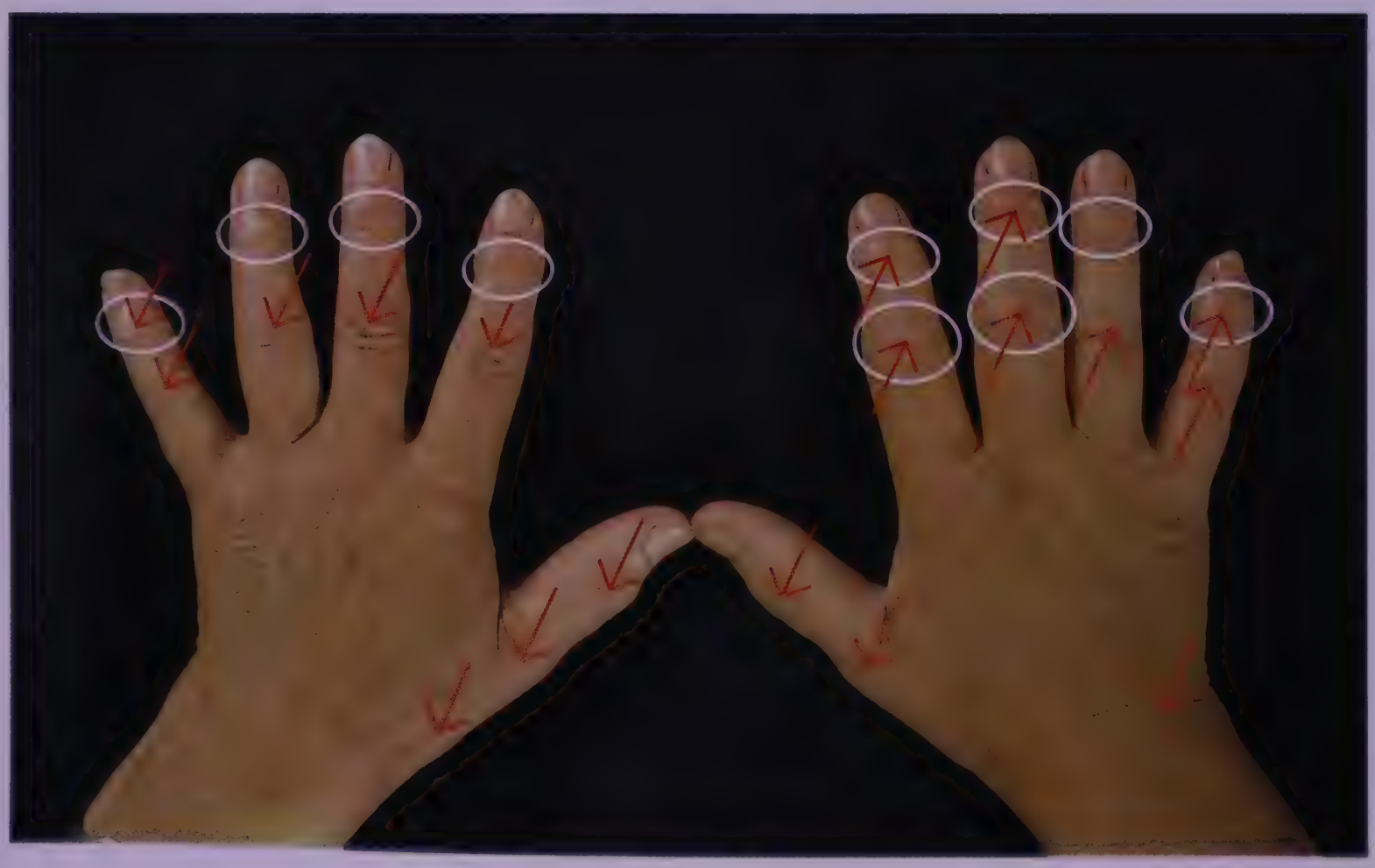
- psoriatic arthropathy
- gout
- pyrophosphate arthropathy

Follow-up

Four years later, at the age of 58, the patient was seen again, with complaints of progressive pain, stiffness, and impaired function of the hands. She was now unable to work. She had been receiving beta-blocker and diuretic therapy for hypertension.

Examination

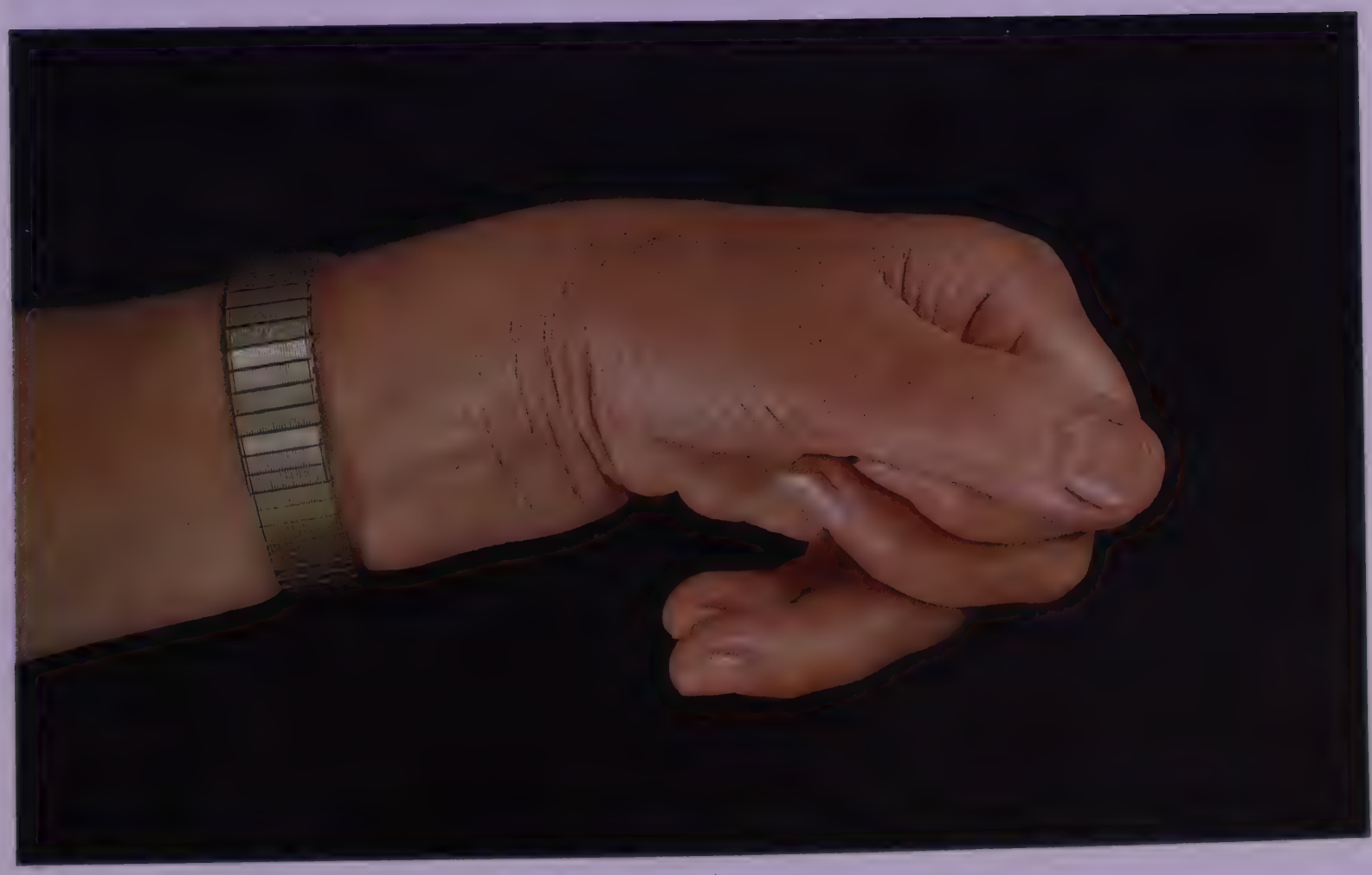
palpable exostoses ↗ tenderness



Functional assessment

The examination revealed tenderness, as well as bone-hard swellings, as illustrated, most of which were not tender. Flexion of most fingers was impaired. She was able to hold a pencil, but the maximum grip strength was diminished to 100 mm Hg on the right side and 80 on the left side (normal levels > 200 mm Hg). This was mostly due to the pain provoked by this exercise.

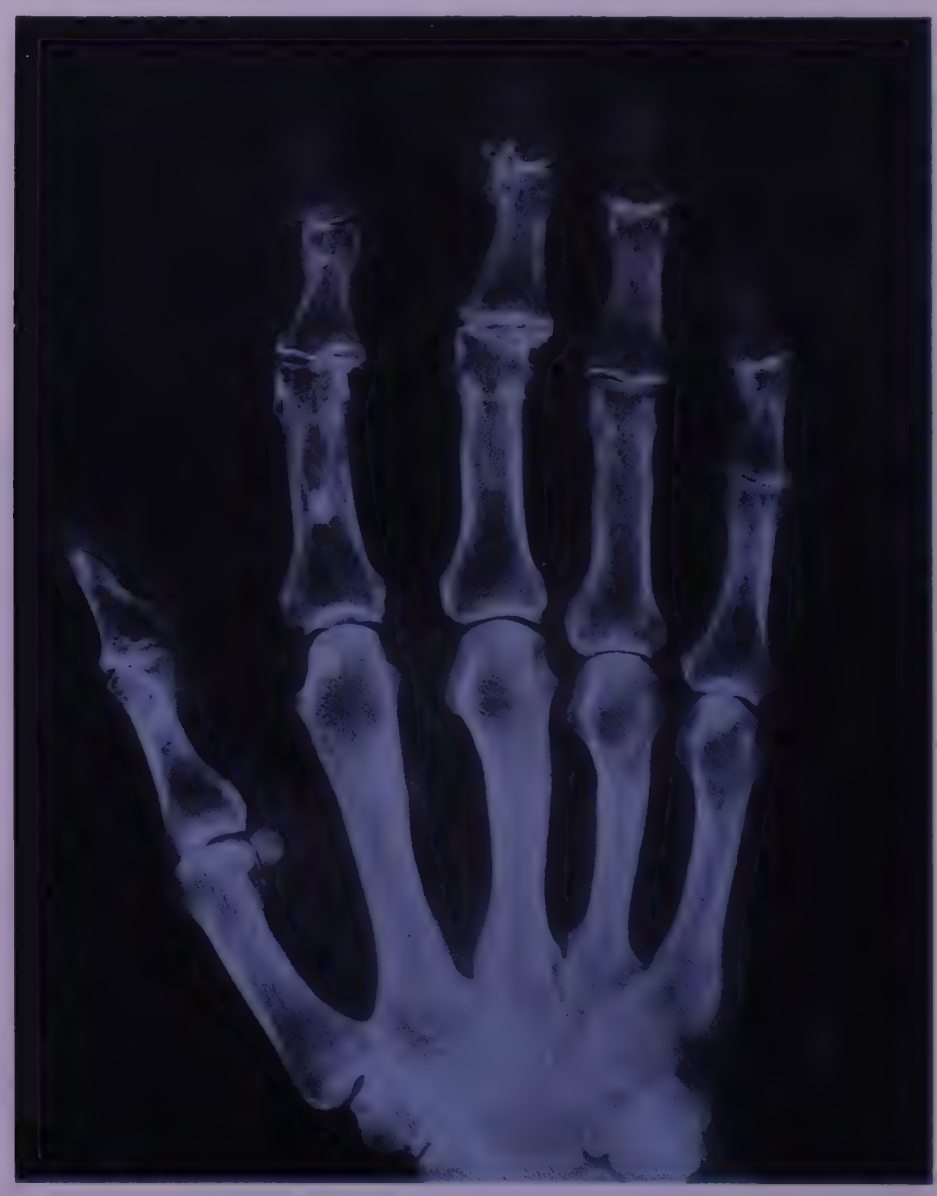
Hand demonstrating maximum flexion of the fingers



Radiography

The X-ray films now showed cartilage destruction and sub-chondral cyst formation of all distal interphalangeal joints. Some of the proximal interphalangeal joints showed similar changes. However, the metacarpophalangeal joints remained intact.

X-ray of right hand



Note: progression of changes in all distal interphalangeal and proximal interphalangeal joints of index and middle fingers.

Management

Attempts were made to reduce her pain by persuading her to try more regular use of NSAIDs. Local glucocorticoid injections were tried with moderate success in two proximal interphalangeal joints. Local heat therapy was also employed. However, for the time being, she was unable to return to work, while still being able to cope with household chores.

Case 2

Mr RB, a 60-year-old divorced shop assistant

"Doctor, I have this intense pain in my fingers. I'm dropping things all the time, and had to give up work 4 months ago."

"When did the problem start?"

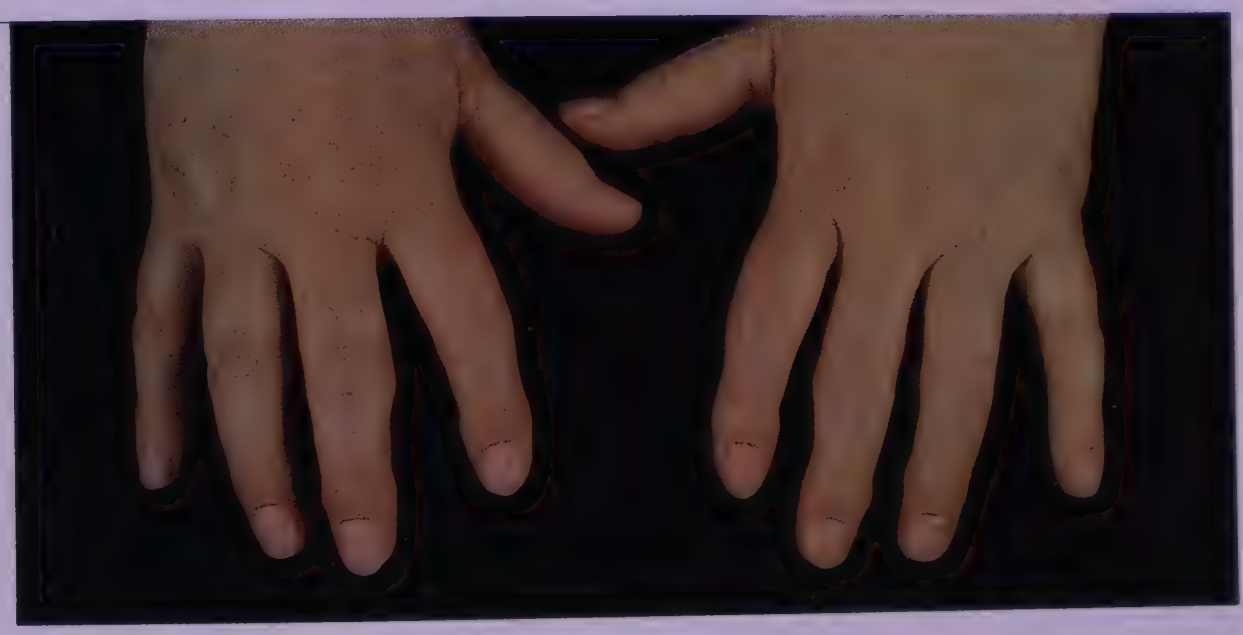
"It started 8 years ago in the outer finger joints, which became swollen, tender, and very sensitive to blows. Then it spread to the other finger joints until I could no longer make a fist. And now I can't bend my fingers properly at all."

"Have you tried any drugs or physiotherapy?"

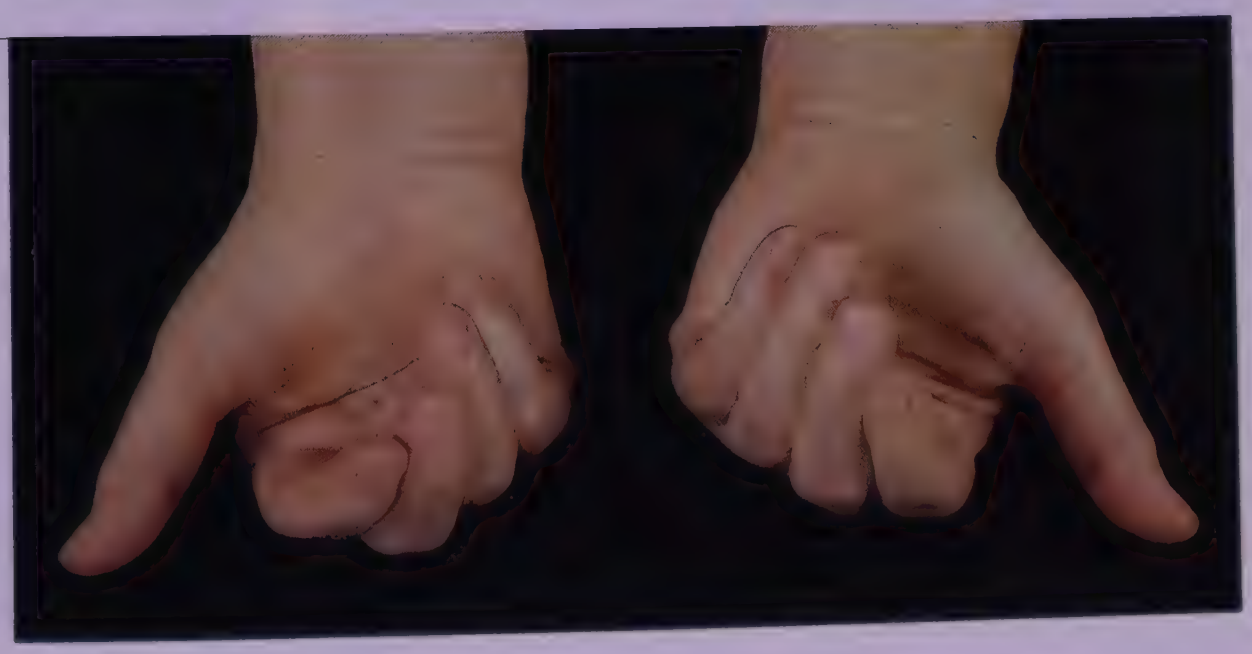
"Aspirin hardly helps, nor does heat. I'm taking ibuprofen which has helped my shoulder pains."

Examination

Moderate signs of supraspinatus tendinitis with pain provocation on outward rotation of the arms against resistance. No other joint abnormalities were found except those in the hands. Inspection of these revealed Heberden's and Bouchard's nodes on all distal and proximal interphalangeal joints. Varying degrees of tenderness were noted over the nodes, as well as markedly diminished active and passive motion, which was intensely painful. No flexion was retained in the interphalangeal joints of the thumbs.



Dorsal view of hands



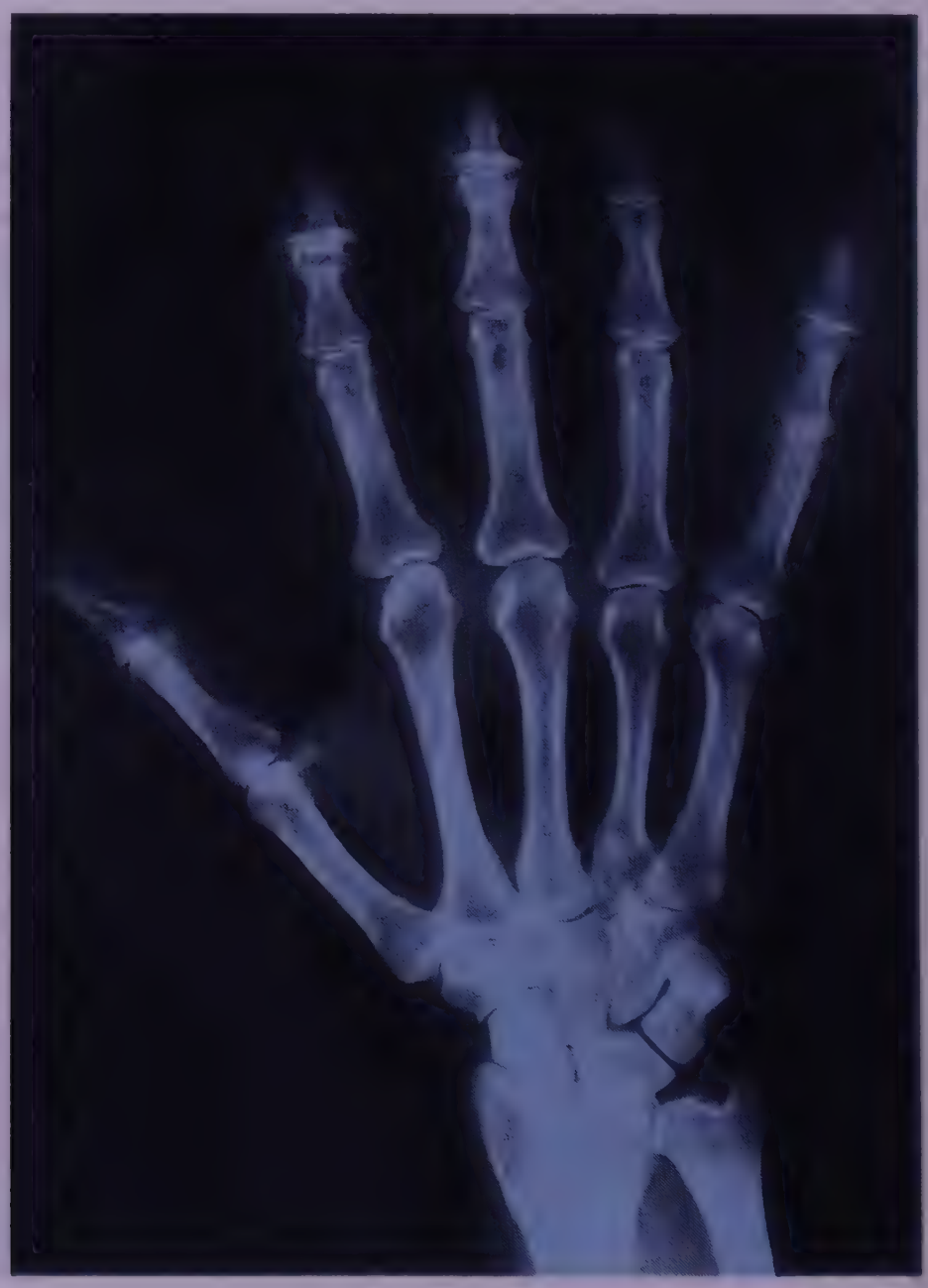
Hands demonstrating fingers in maximally flexed position

Investigations

The sedimentation rate was only 4 mm/h. The rheumatoid factor test proved negative. Haemoglobin, uric acid, serum calcium, and alkaline phosphatase were normal. The radiograms showed advanced cartilage destruction, joint surface irregularities, subchondral cysts of the bones, and exostoses.

Radiological progression 1980–1988

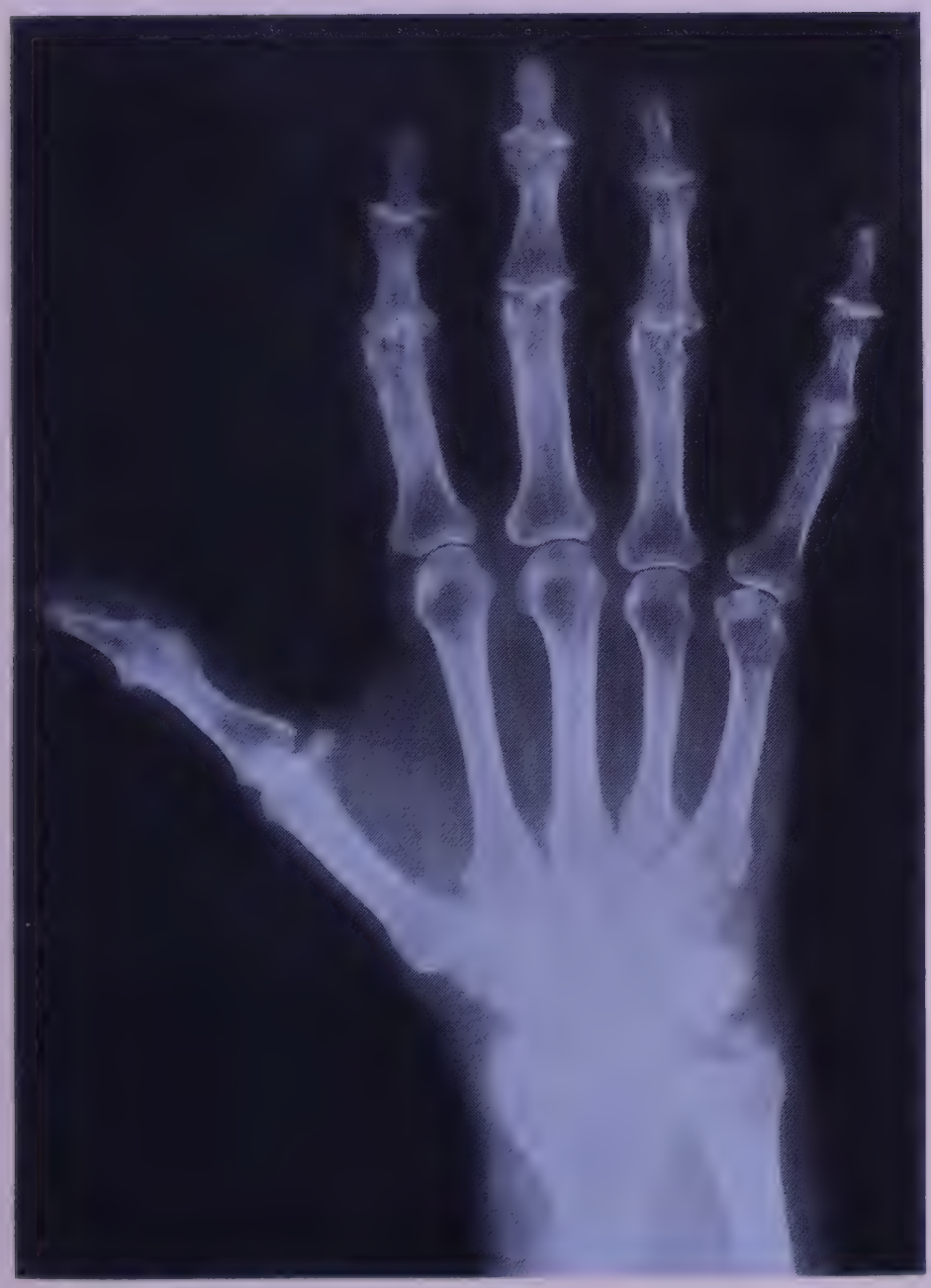
a) Cartilage destruction and bone erosions of distal interphalangeal joints, combined with pronounced osteophytosis. Typical "bird's wing" of DIP V.



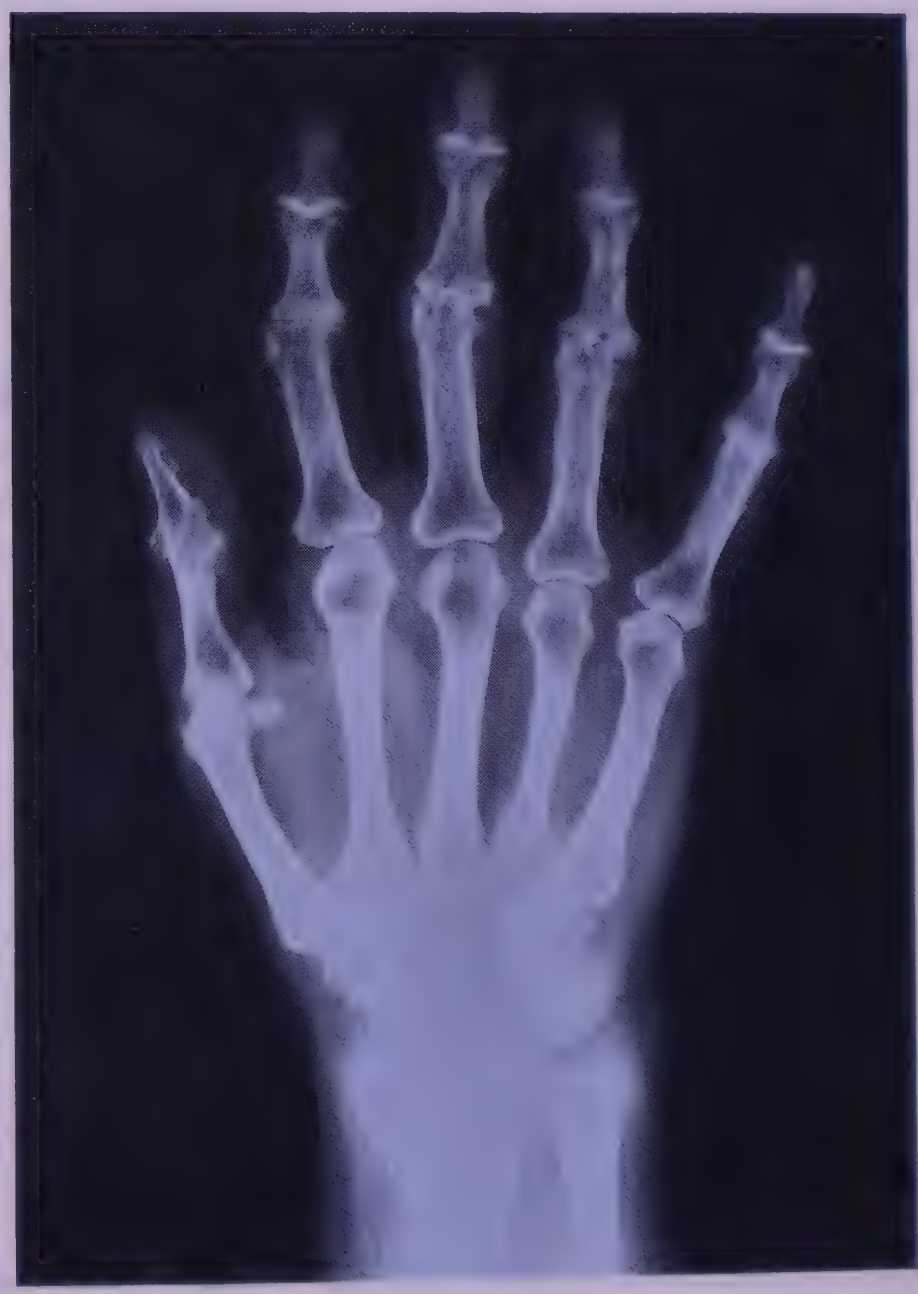
a)

b) Similar changes now also seen in proximal interphalangeal joints. Destruction and bony hypertrophy of first interphalangeal joint.

c) Progression of subchondral cyst formation and osteophytosis. Note absence of osteoporosis and intact metacarpophalangeal joints.



b)



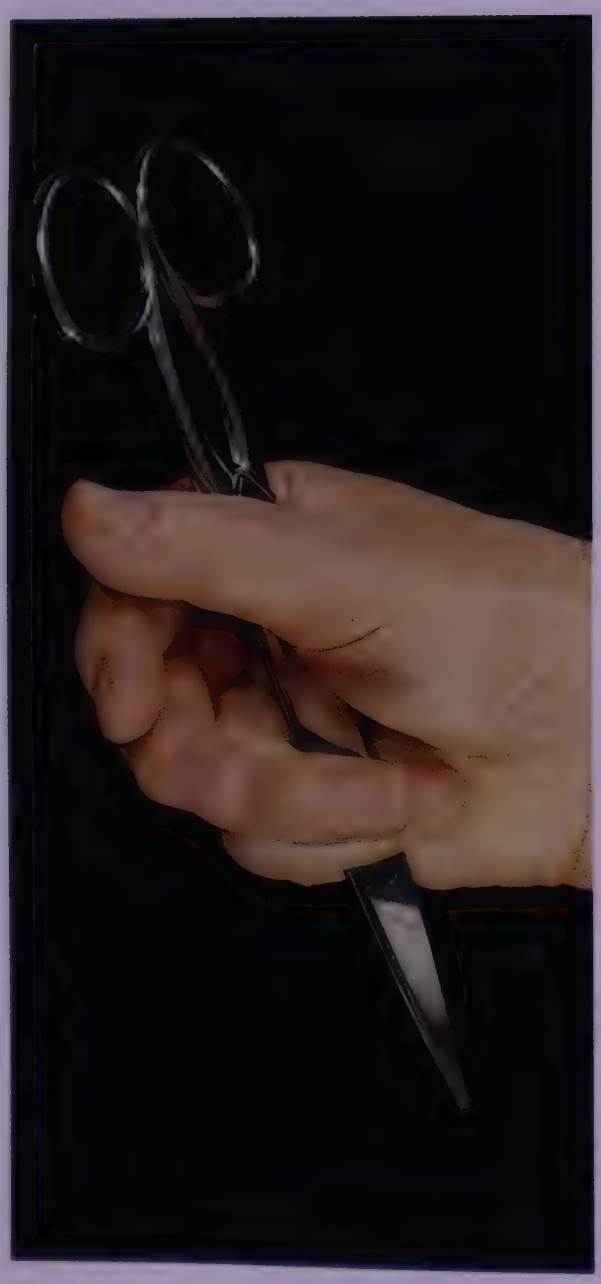
c)

Management

Switching to another NSAID improved pain at rest, but due to continued pain on motion and the restricted range of motion a hand surgeon was consulted. It was agreed that arthrodesis of several proximal and distal interphalangeal joints would be the best treatment in this advanced case of destructive osteo-arthritis.

Follow-up

After surgery the patient experienced complete relief of pain in all operated joints. He could still not make a closed fist, but he could use his hands more and was able to return to part-time work.



Postoperative – pain-free and functioning

Case 3

Mrs EB, a 50-year-old nurse, who had undergone retraining and become a secretary two years ago because of low back problems

"Doctor, I like my new work very much, but over the last months I've been experiencing really sharp pains in my fingers."

"Is it a constant pain?"

"No, it comes and goes, and sometimes the fingers become red and hot."

"Any joint problems in your family?"

"My mother had fingers that were all crooked, and they often hurt as well."

Examination

The distal interphalangeal joints of both hands showed discrete swellings of nodular (Heberden) type. Three were also reddened and tender on palpation. Grip strength and flexion was intact. She also had pain on inward rotation of the hips, and some crepitations could be felt.

Investigations

Radiological signs of cartilage destruction in the distal interphalangeal ring and little fingers were present, and a few small osteophytes could be seen. ESR was 5 mm/h, haemoglobin 125 g/L. Normal uric acid and negative rheumatoid factor.

Diagnosis

The patient was told that she was suffering from osteoarthritis and that the prognosis was good. She was given a prescription for a NSAID, which was to be taken intermittently, when pain became a problem.

Follow-up

This patient was seen again 12 and 16 years later, when she underwent total hip surgery for osteoarthritis. She had been working full time until her retirement at age 65. She now had marked Heberden's and moderate Bouchard's nodes, but had normal hand function and no tenderness.

Characteristic features of osteoarthritis (OA) of the upper extremities

Localisation:	
	Shoulders: Acromioclavicular joint
	Elbows: Uncommon, mostly secondary
	Hands: Trapezius-scaphoid (TS) joint First carpometacarpal (CMC) joint Proximal interphalangeal (PIP) joints (Bouchard's) Distal interphalangeal (DIP) joints (Heberden's nodes)
Nodal type:	
	Heberden's, women, familial
Non-nodal:	
	Bouchard's, men *and* women
	Inflammatory episodes, obesity
Erosive OA:	
	Postmenopausal women, PIP and DIP joints, inflammatory episodes, pannus and synovial infiltration, negative rheumatoid factor

Aetiology

Genetic factors are of some importance but their mechanism is unknown. Crystal deposition is strongly suspected to be a major pathogenetic factor. Calcium pyrophosphate and calcium apatite crystals are often seen when looked for. Trauma with overload and autoimmune mechanisms have been suspected.

Prognosis

Although OA lesions are often progressive and rarely heal, symptoms in the upper extremities, and in particular in finger joints, are usually of a transient nature. This is not so, however, in the erosive form, represented by case 2.

Differential diagnosis

Psoriatic arthritis: younger age, skin involvement, nail changes in 80% of cases, elevated sedimentation rate and uric acid.

Tophaceous gout: aspiration of tophi and examination by polarised light microscopy.

Rheumatoid arthritis: synovitis, absence of palpable osteophytes, localisation, elevated sedimentation rate, radiological evidence of osteoporosis, erosions at preferential sites, predominance of proximal interphalangeal joints and metacarpophalangeal joints, ulnar deviation, buttonhole and swan-neck deformity, muscle atrophy.

Management

1. The patient should be informed of the established diagnosis and prognosis.
2. Pain relief by adjusted load, heat, and non-salicylate analgesics, i.e. paracetamol and NSAIDs.
3. Local injection of glucocorticoids when signs of inflammation are present. Repeat if effective, but not more than every 3 months.
4. Splinting, in particular of first carpometacarpal and interphalangeal joints.

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Osteoarthrosis of hip and knee

J.K. van der Korst

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Case 1

Miss Johnson, an 18-year-old student, walks into the consulting room.

"My right knee has been causing me trouble over the last few months, especially when I walk down stairs. The pain has become a nuisance when I'm cycling and playing hockey, especially in the last two weeks."

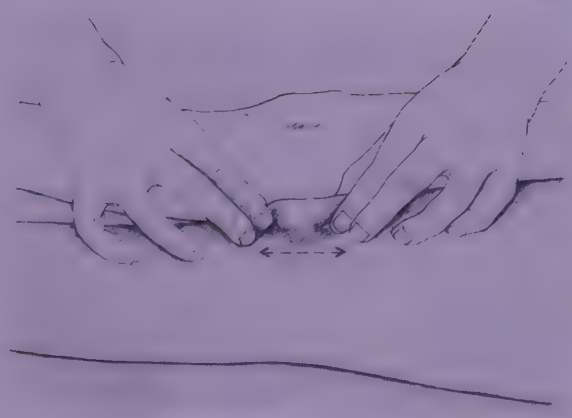
"Did you hurt your knee in some way?"

"No, not that I can remember."

"Has the knee been swollen for some time?"

"No, the knee has always looked perfectly normal, as far as I can see."

"I'd like to have a look at both your knees. Would you take off your shoes and trousers, please."

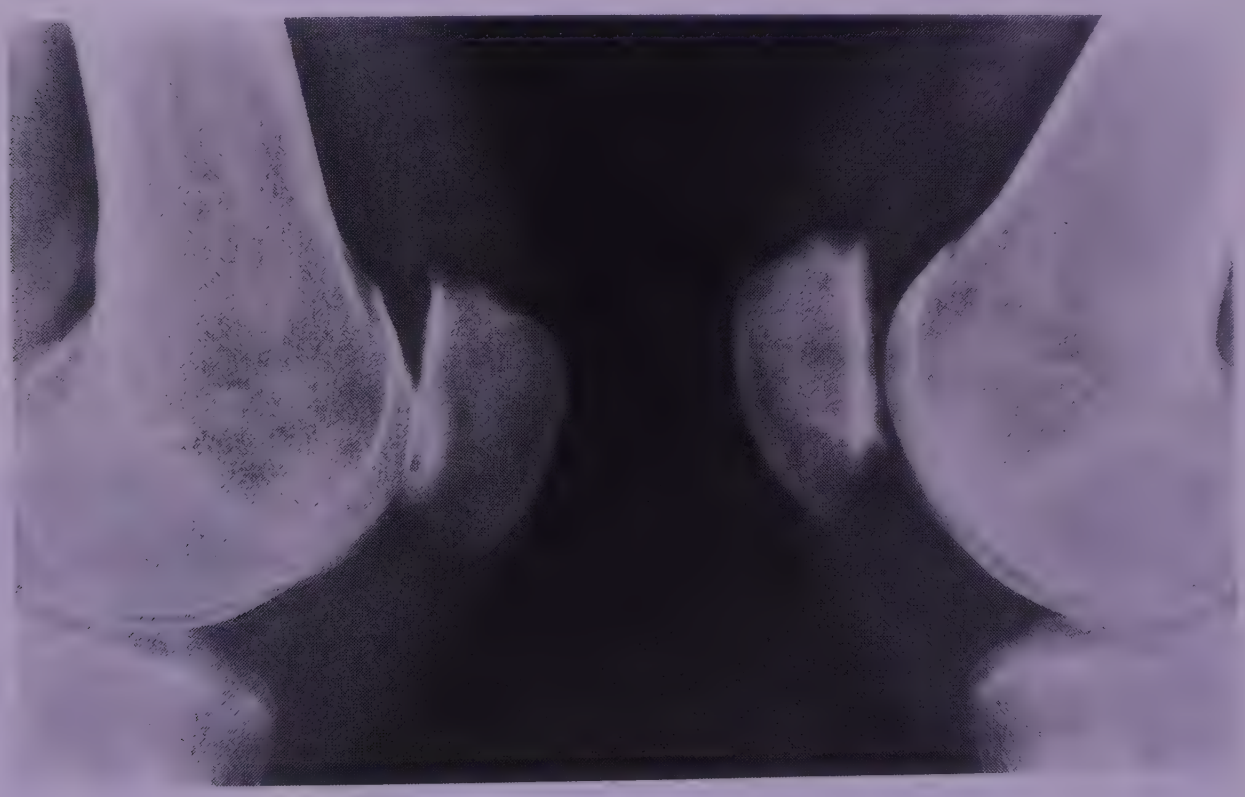


Examination

Indeed, the knee looked perfectly normal. Neither did it feel warm, and no sign of hydrops could be detected. However, when the knee was passively flexed, the patient indicated pain in the patellar region. When the leg was put down again and the patella was moved over the femoral condyles, crepitations could be felt, and the patient again indicated pain.

Radiology

There was clearly an indication for X-ray examination of the knee joint and, especially, of the patellofemoral joint. Although the diagnosis of chondromalacia patellae or (early) patellofemoral osteoarthrosis was sufficiently clear from the patient's presenting signs and symptoms, radiology was needed to establish the degree of degenerative change that had already occurred.



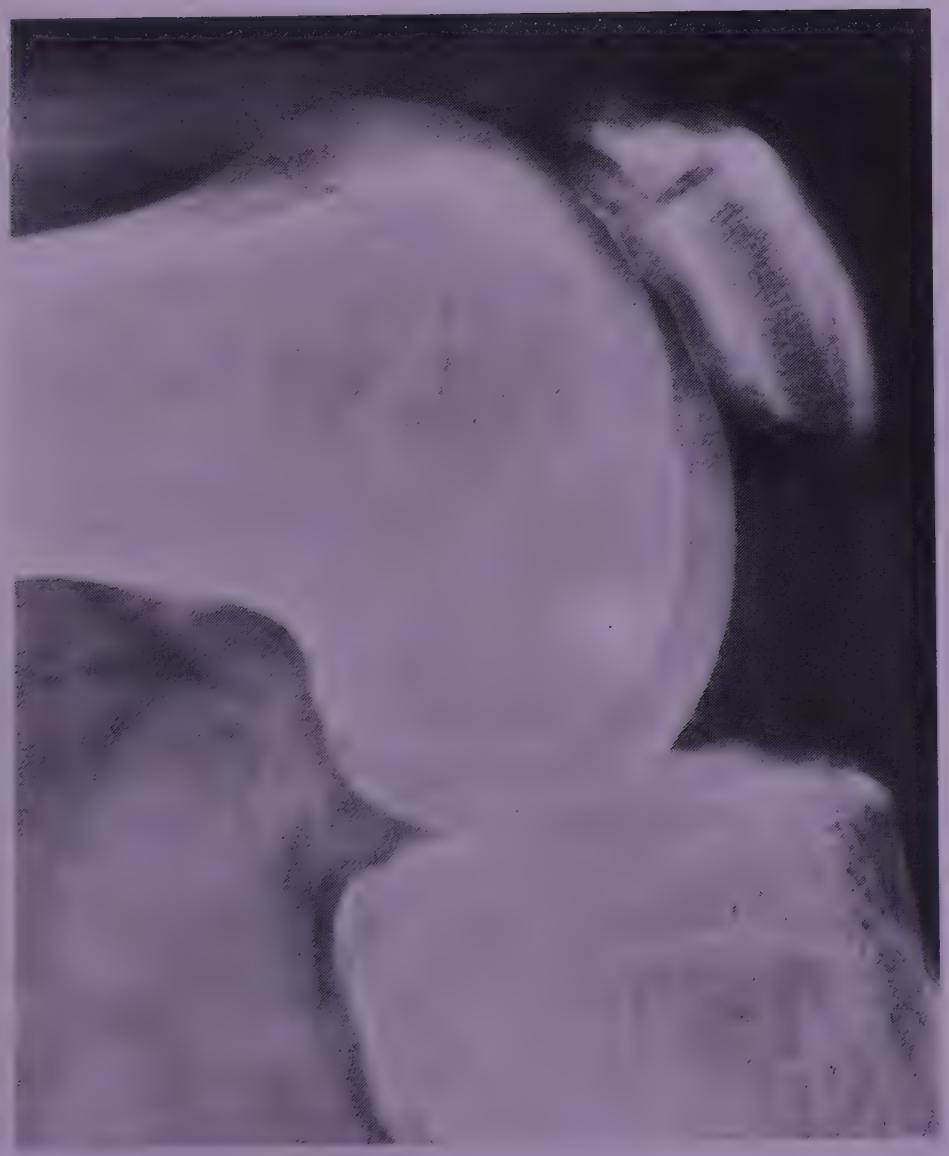
Differential diagnosis

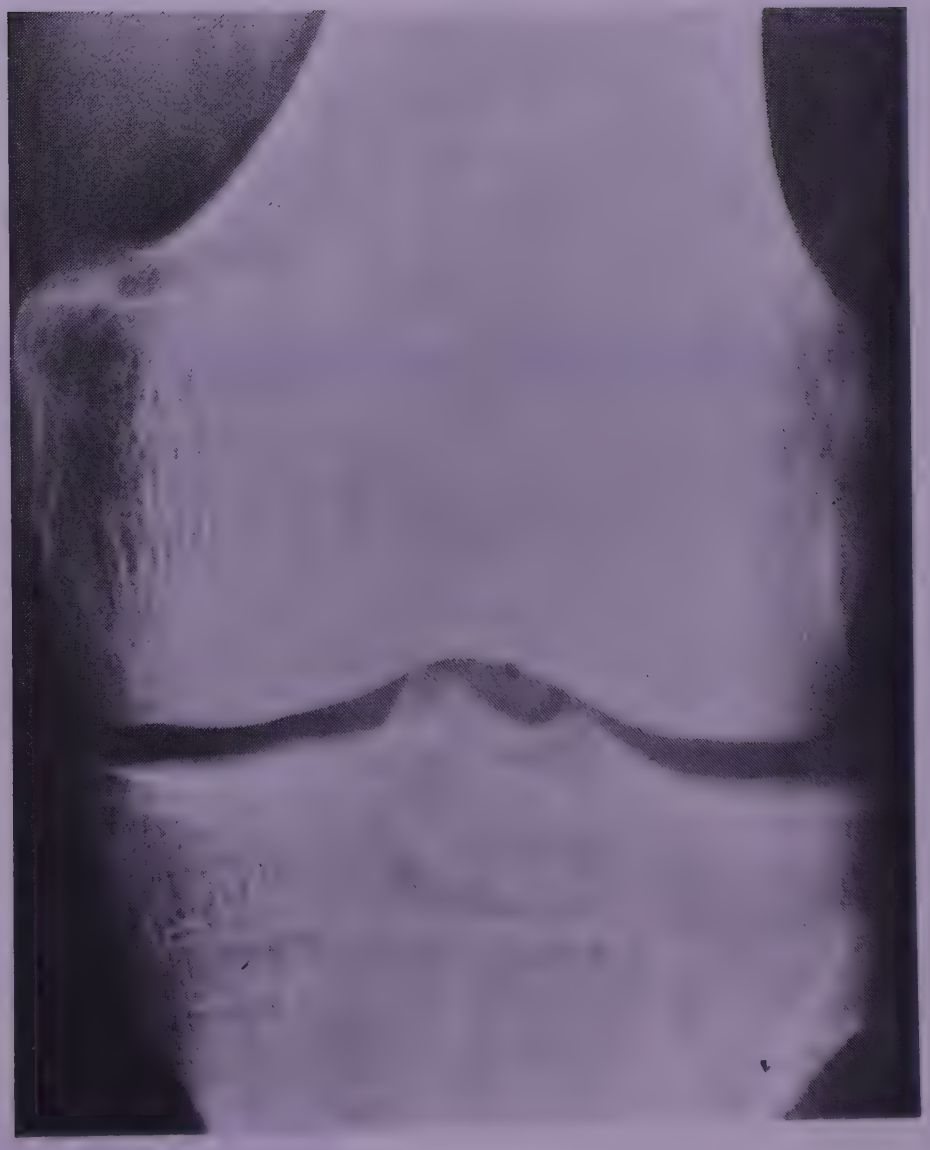
Chondropathy of the patella, a seemingly frequent condition in adolescents, may be due to a variety of underlying conditions, although in most cases it is "idiopathic". It is essential to try and ascertain whether there is such an underlying cause, since this may go a long way in helping to assess the prognosis and sometimes the necessary therapy as well.

1. Traumatic knee instability

Chondromalacia patellae may be the first and only sign of torn ligaments. Therefore, examine both the lateral and the anteroposterior stability of the knee. Surgical treatment might be indicated.

2. Congenital dysplasia of the patella,
for instance a bipartite patella. Only radiology will establish the abnormality in most cases. Sometimes, surgery is indicated.





3. Generalised hypermobility

Generalised hypermobility – also known as "joint laxity" – predisposes to polyarticular osteoarthrosis. In most cases patellar complaints are the first hallmark of this condition. Establish whether the knee is able to extend more than 15 degrees and look also for the range of motion of other joints (the figure illustrates examination of the hand), as well as of the spine.
In the case of Miss Johnson the X-rays of the knee and the patella revealed no abnormalities. The stability of the joint was perfectly normal and there were no signs of hypermobility whatsoever.

"Miss Johnson, there are only signs of some overstressing of your kneecap, which have resulted in a roughening of the cartilage. This is quite common at your age. We only have to take care that your knee gets some rest. I will refer you to a physiotherapist, who will instruct you as to how you should protect your kneecap from overstressing, and he will also see to it that your leg muscles are trained in the right way."

Diagnosis: Idiopathic patellar chondropathy

Case 2

Mr. Peters, a 45-year-old physiotherapist, asks your help in a personal matter which seems to be preying on his mind.

"During the last few months I have noticed something wrong with my right hip. It becomes increasingly painful when I'm walking and standing. I am afraid it might be some form of arthritis."

"Is it only your right hip that is bothering you?"

"Yes. Otherwise my joints are all right, and I feel perfectly well. About six months ago – during a holiday – I suddenly got urticaria all over my body. It disappeared during a course of prednisone, which my travel companion – a doctor – gave me."

"I would like to examine you."

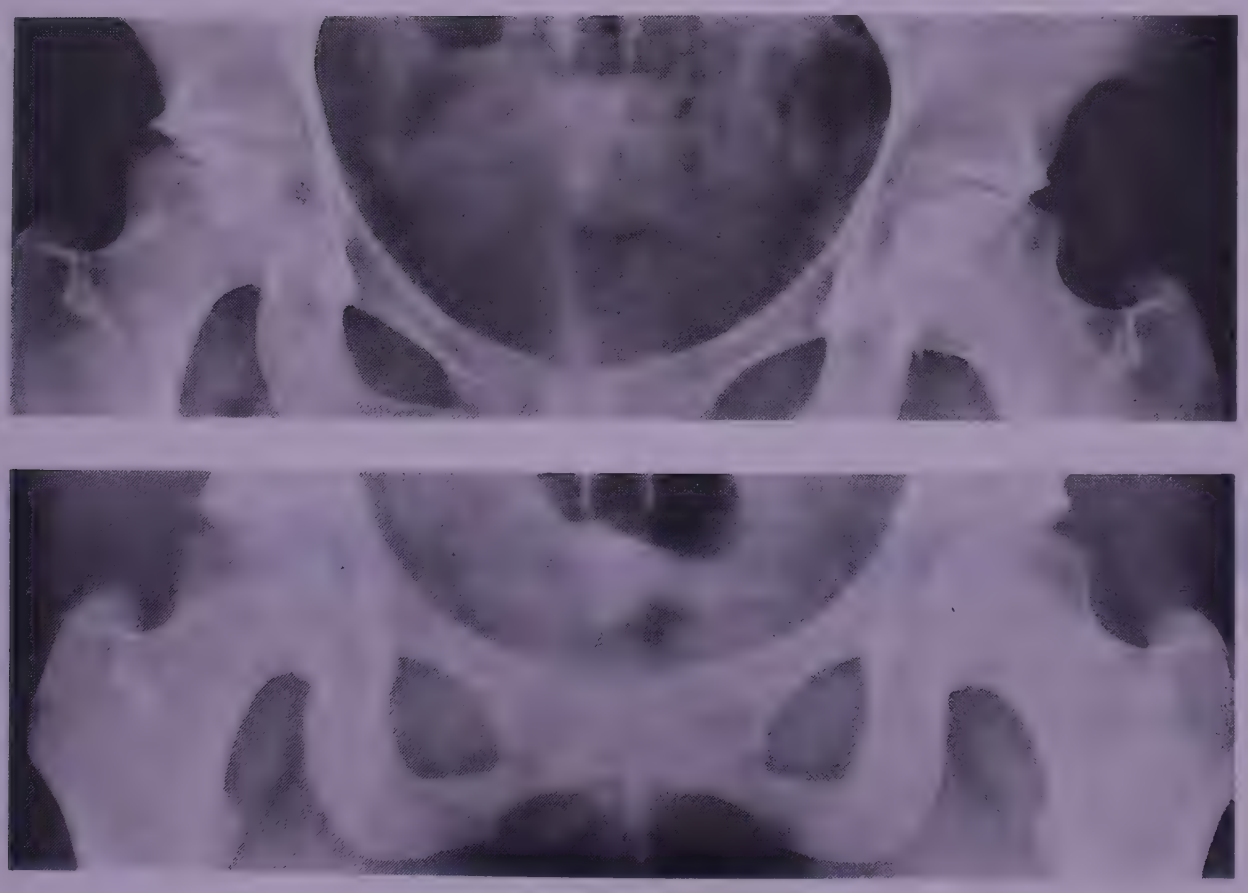
Examination

At physical examination the only abnormalities that could be found were rather slight limitations in the mobility of the right hip, especially on inward rotation.

"Well, it is quite clear that something is wrong with the hip joint. At this stage it is impossible to say whether it is some form of arthritis or osteoarthrosis. In the first place we need to know the sedimentation rate of your blood, and we will take some X-rays."

Additional subsequent findings

The ESR turned out to be normal: 7 mm in the first hour.
The radiogram of the pelvis showed a flattening of the upper pole of the right femoral head and a translucency of the underlying bone structure.



Diagnosis: Aseptic necrosis of the femoral head

Aseptic bone necrosis

Subchondral bone necrosis may be considered a kind of ischaemic infarction of the epiphysis. It most commonly occurs in:

- the femoral head
- the navicular bone of the carpus
- a femoral condyle
- the humeral head.

A large variety of factors may be involved in the pathogenesis of aseptic bone necrosis, but the most frequent are:

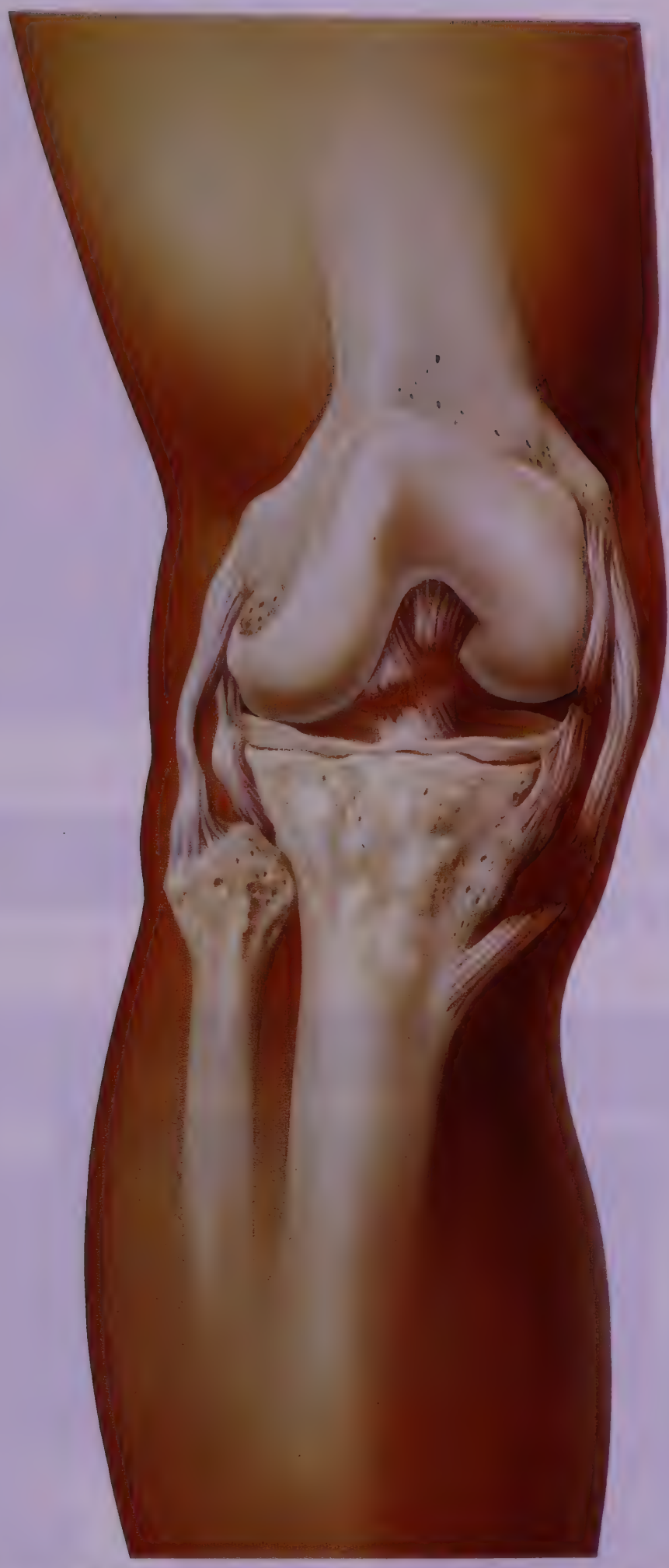
- corticosteroid usage
- alcohol abuse
- hyperuricaemia.

Sometimes, but not always, aseptic bone necrosis causes symptoms during the acute phase. Otherwise, it might eventually lead to osteoarthrosis of the nearby joint due to deformity of the epiphysis.
Aseptic bone necrosis is one of the many causes of **secondary osteoarthrosis,** such as arthritis, congenital dysplasia, recurrent joint bleeding, chondrocalcinosis, etc.

"Mr. Peters, your hip trouble is caused by a disturbance of the blood flow in the femoral head; this has probably been provoked by the cortico-steroids that you have been taking. As a result, part of the bone structure is dead, but new bone will be formed. In the meantime, the femoral head is weak, and there is a serious danger of deformity which might result in osteoarthrosis of the hip. I would urge you to spare your hip as much as possible during the coming months."

Subsequent clinical course

The complaints of the hip subsided slowly during the following months, but after one year a progressive stiffness occurred, as well as increasing pain while walking and standing. Severe degenerative changes became demonstrable radiologically and after five years arthroplasty became necessary to enable the patient to continue his practice.



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1 Van Heerden, J.J. Diclophenac Sodium, Oxyphenbutazone and placebo in sports injuries of the knee. S.A. med J. 52 (10): p. 396-399, 1977.

Case 3

You are paying a visit to Mr. de Woolf, a 70-year-old retired farmer, in whom you diagnosed pneumonia a few days earlier, and who is now being treated with an antibiotic.

"How are you getting on now? You look somewhat better."

"Yes, I really do feel somewhat better. But this morning I woke up very early because of my left knee, which hurts so badly. I dare not move it at all."

"When did the pain start?"

"Last night. Yesterday the knee still felt all right. You know, it is my bad knee."

History

Indeed, you know Mr. de Woolf has osteoarthrosis of the left knee, which started years ago after he fell from a horse. Most of the time, this condition has hardly bothered him at all until now.

"Let me have a look at your knee."

Examination

The left knee appears acutely inflamed: it is red, swollen, and hot. The patient is unable to move, and even the slightest touch is very painful.

Differential diagnosis

When an osteoarthritic joint shows signs of acute inflammation, one should first consider the following three possibilities:

1. Septic arthritis.
Joints suffering from osteoarthrosis run an increased risk of being infected in the course of a bacteriaemia.

2. "Activated" osteoarthrosis.
Osteoarthrosis is very frequently accompanied by signs of joint inflammation due to cartilage fragments and abnormal products of chondrocyte metabolism reaching the synovial space. Acute "activated" osteoarthrosis is often caused by "overusage" of the joint.

3. Pseudogout.
In older patients osteoarthrosis is frequently accompanied by chondrocalcinosis, i.e. "calcification" of joint cartilage indicating deposition of calcium pyrophosphate. Under circumstances of physical stress, e.g. surgery or infectious disease, "crystal shedding" in the synovial space might occur, giving rise to an acute (gout-like) arthritis: pseudogout.

"Well, it does not seem serious to me, but I have to know for sure that your knee is not infected by the same germs which have caused your pneumonia. Therefore I have to puncture your knee."

Joint puncture

Take a sterile needle and syringe and put on plastic gloves. Sterilise the skin. Puncture the knee at the spot where fluctuation is most readily demonstrable.

Examine the obtained fluid for:

- appearance
- colour
- viscosity.

Eventually the joint fluid may be cultured or examined for crystals.

"What I got out of your knee is clear. So, we don't have to bother about infection. I will prescribe you an antirheumatic drug, and in a few days the knee should be much better."

Subsequent clinical course

Indeed, within a few days the signs of inflammation of the knee joint disappeared during treatment with a non-steroidal anti-inflammatory drug. At your office you find the report of the latest X-ray of the knees, stating that besides degenerative changes the left knee also shows chondrocalcinosis (Figure). Moreover, the aspirated joint fluid, examined by the pathologist, was found to contain numerous birefringent rod-like crystals, compatible with chondrocalcinosis.

Diagnosis: Pseudogout

Pain in osteoarthrosis

Osteoarthrosis is primarily a degenerative condition of the joint cartilage. Cartilage does not contain nerves or nerve-endings. Therefore cartilage itself can never hurt. Nevertheless, osteoarthrosis is frequently accompanied by pain, and often pain is the first sign of degenerative joint disease.
There are three different mechanisms by which osteoarthrosis might induce pain:

1. Abnormal stretching of ligaments.
This is especially marked in cases of patellofemoral osteoarthrosis and may occur at a very early stage. One of the first consequences of "roughness" of the joint cartilage is an increased friction when two cartilage surfaces are gliding over each other. The resulting restistance will induce abnormal stretching forces of the involved ligaments causing a proprioceptive pain response.

2. Synovial inflammation.
This is probably the most common cause of pain in osteoarthrosis. In osteoarthrotic cartilage the metabolism of the "exposed" chondrocytes is disturbed, leading to the formation of crystalline products, such as pyrophosphate and hydroxyapatite. These crystals might "irritate" the synovial membrane.

3. Increased subchondral bone pressure.
This will occur only in cases of advanced osteoarthrosis. Due to the loss of the protective function of the degenerated cartilage, the underlying bone is subjected to "abnormal" compression and friction forces. Consequently many lesions will occur in the bone – sclerosis, for instance. These are eventually accompanied by changes in the vascularisation of the bone and increased intraossal pressure, producing a constant "nagging" pain, which worsens during loading of the bone.

Principles of management in osteoarthrosis

1. Joint protection

As long as osteoarthrosis has not advanced to an almost complete loss of joint function, the most important measure is to protect the joint from unnecessary loading and friction.
In the case of hip and knee osteoarthrosis this objective is best achieved by means of:

- a walking aid
- bandaging of an unstable knee
- avoiding unnecessary standing
- training atrophic muscles etc.

2. Administration of a non-steroidal anti-inflammatory drug

As stated, synovial inflammation is an important cause of pain in osteoarthrosis. Moreover, inflammation will contribute to the degeneration of the articular cartilage. Therefore, treatment with a non-steroidal anti-inflammatory drug is indicated in the majority of cases.
However, since potent NSAIDs might also interfere with pain as a warning sign for "overloading" of a diseased joint, the dosage should not be too high, and at the same time the patient should be carefully instructed with regard to joint protection!

3. Joint surgery

Although arthroplasty is only indicated where joint function has been seriously impaired, in some cases of secondary osteoarthrosis of hip and knee, corrective surgery might prevent the progression of degenerative lesions at an earlier stage.

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Rheumatology in everyday practice

Arthritis

W. Irniger and T. L. Vischer

Vol.3

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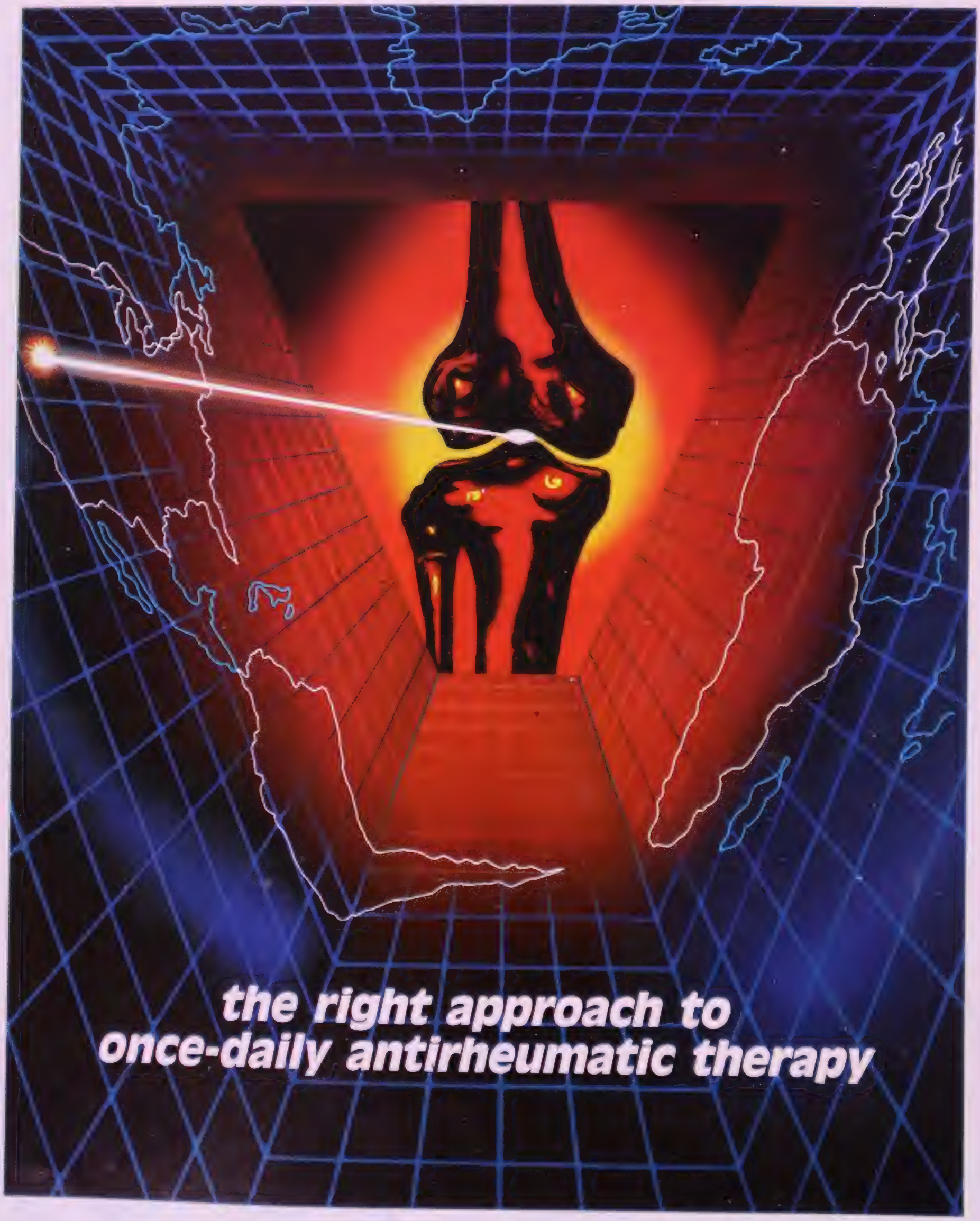

Arthritis

W. Irniger

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Acute arthritis

Differential diagnosis of acute monarthritis

1. Osteoarthritis flare
2. Microcrystal-induced arthritis: gout or pseudo-gout
3. Arthritis at the beginning of a rheumatic condition: e.g. rheumatoid arthritis
4. Reactive arthritis
5. Septic arthritis

A similar clinical picture may also result from traumas, bone lesions (including tumours), or algodystrophy.

Case 1:

Mr. Miller, a 55-year-old farmer, limps into the consulting room.

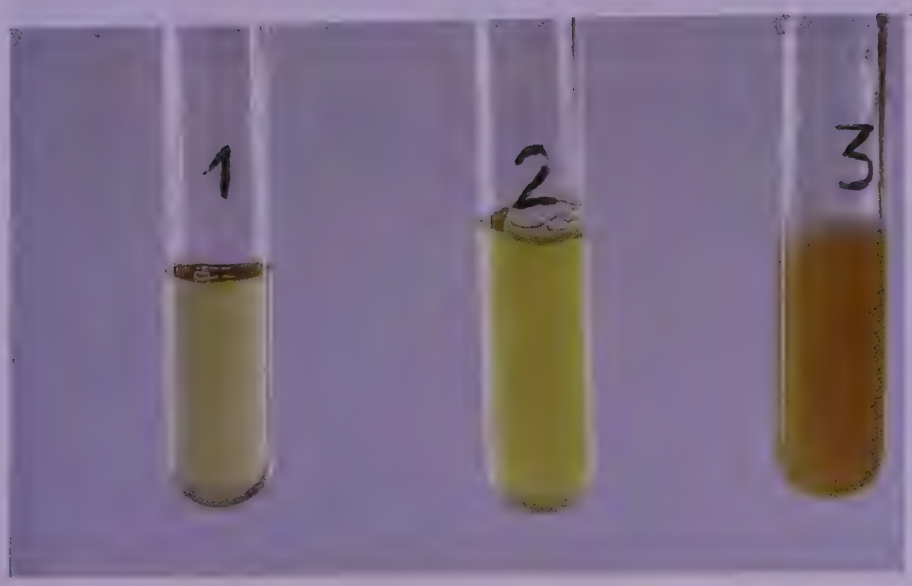
"My right knee has been giving me pain for the last three months, and now it's also swollen."

"Have you ever had knee trouble before?"

"No, not really – except that, after I'd been walking downhill for quite a distance, I sometimes used to feel a bit of pain; and now and then I also used to find the first few steps a little painful the next morning."

Examination

The right knee joint shows marked blunting of the contours, but the skin is cool; although the knee is not particularly tender, effusion is clearly apparent upon palpation. An exploratory aspiration yields approx. 30 ml clear yellowish synovial fluid.



Synovial fluid: clear fluid contains only a few cells and is indicative of only very mild irritation. In the first tube the cell count was 300 per mm^3 (sprain), in the second tube 5,900 per mm^3 (Reiter's syndrome), and in the third tube 150,000 per mm^3 (septic arthritis). Leucocyte counts of up 100,000 cells per mm^3 may also be found in cases of rheumatoid arthritis or microcrystal-induced arthritis.

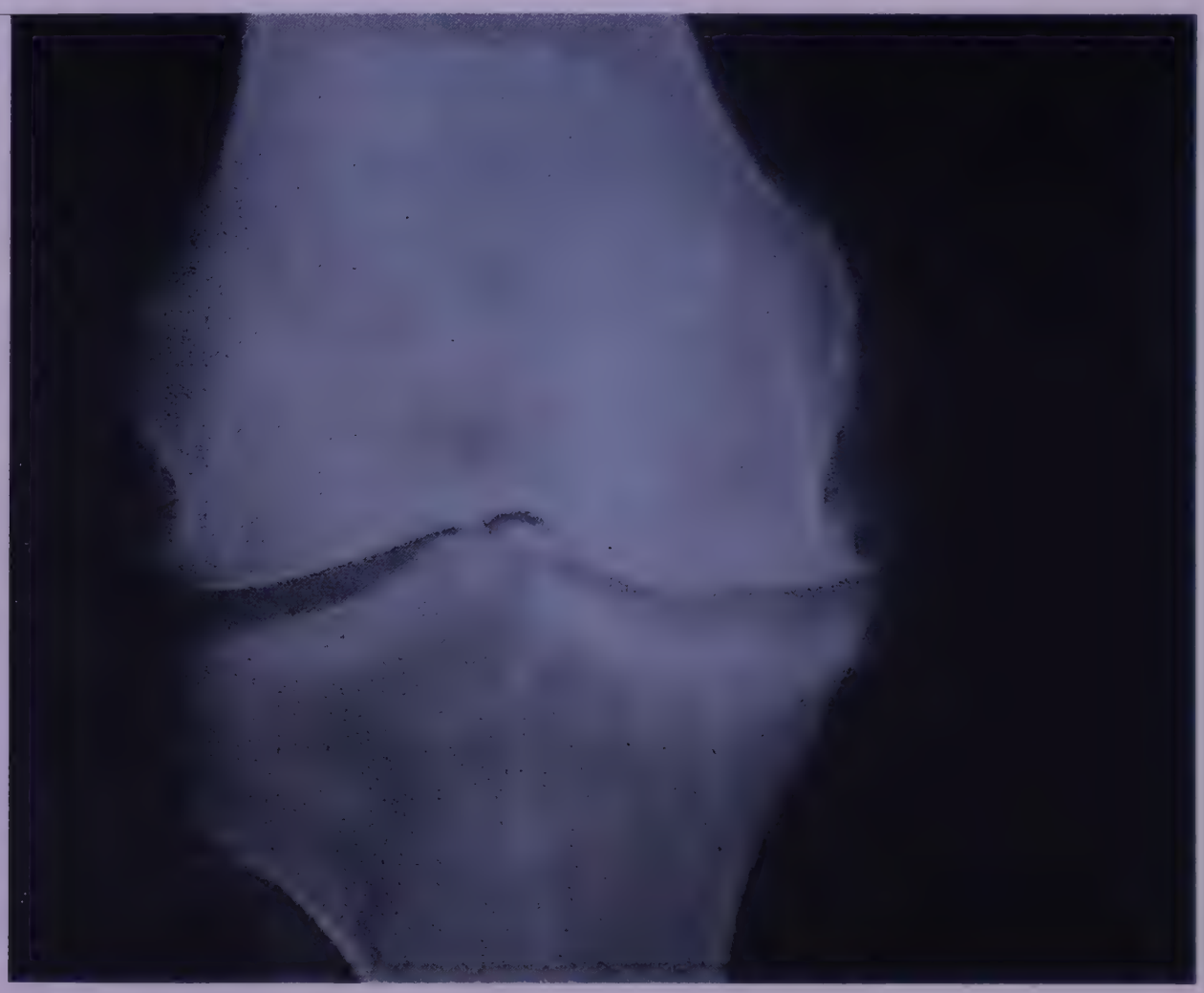
Viscosity

Left: urate crystals; they are needle-shaped and usually present in large numbers. Right: calcium pyrophosphate crystals; they are shaped rather like beams and are usually less numerous. Crystals can best be identified under polarised light.

Examination of synovial fluid

1. Appearance	
	Clear (few cells)
	Cloudy (many cells)
2. Colour	
	Red (blood)
	Yellow (reactive effusion)
	Whitish (many cells)
3. Viscosity	Allow one drop to drip from the syringe.
	If the drop forms a thread:
	high viscosity
	(reactive effusion)
	If it forms no thread:
	reduced viscosity
	(inflammatory effusion)
4. Culture	Do not forget the possibility of tuberculosis.
	Monarthritis should always arouse the suspicion of infection. If the joint has already sustained damage (rheumatoid arthritis or osteoarthritis), or if the patient is old, the clinical signs of an infection may often be inconspicious.
5. Examination for microcrystals	
	Urate crystals permit a diagnosis of gout, and calcium pyrophosphate crystals a diagnosis of pseudo-gout (chondrocalcinosis).

Since the farmer's house is a long way from the practice, to spare him the inconvenience of having to pay another visit the knees are X-rayed now; the radiograph shows incipient osteo-arthritis (OA).



OA of the knee:
joint-space narrowing (especially of the medial compartment), sclerosis, and osteophytosis.

"Mr. Miller, your trouble's due to irritation of the knee joint, which is beginning to show a few signs of wear and tear. I'll give you some medicine now to ease the pain; but, even when the knee feels all right again, you'll have to take good care in future not to put too much strain on it. In other words, you should not carry heavy loads, especially when walking downhill. You should also wear soft, rounded rubber heels. Please let me know how things are progressing in a couple of week's time."

Diagnosis: OA flare

Indications for X-ray examination in fresh cases of single joint involvement

1. To exclude the possibility of bone lesions, e.g.

- Post-traumatic
- Osteochondritis – aseptic necrosis
- Tumours
- Algodystrophy.

2. For the diagnosis of osteoarthrotic lesions

Narrowing of the joint space (in the case of the knee joint, preferably during weight-bearing, i.e. standing)

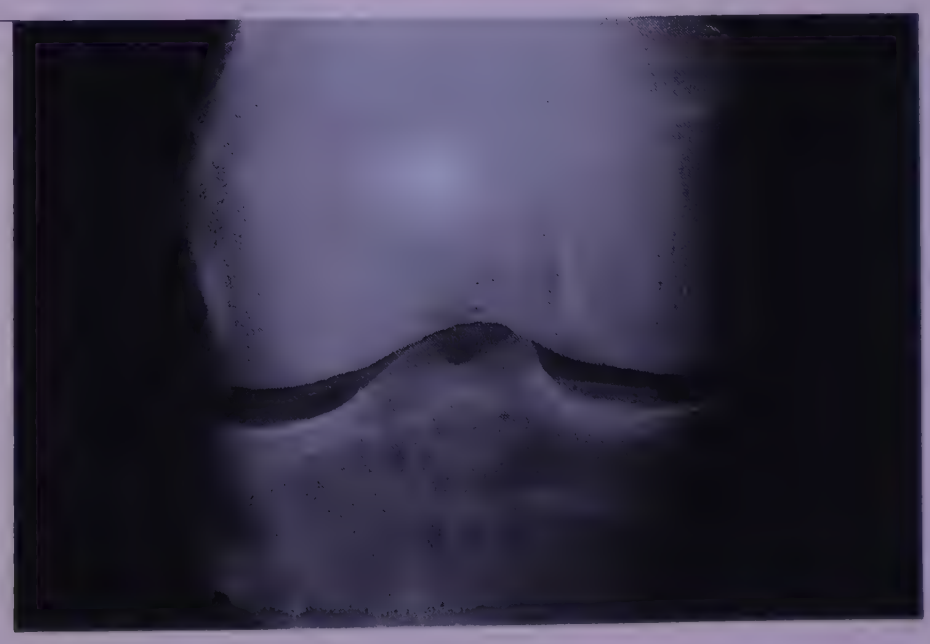
- Sclerosis
- Osteophytosis
- Chondral debris cysts.

3. For the diagnosis of chondrocalcinosis

Since OA may assume a latent (i.e. clinically silent) form, attempts should be made to establish concordance between the X-ray and the clinical findings.

Where an X-ray examination is done, the contralateral joint should also be X-rayed in order to establish anatomical variations which are usually symmetrical.

Chondrocalcinosis: meniscal contours showing typical calcification.



Two weeks later, Mr.Miller telephones to say:

"As soon as you had removed the water from the knee, it felt fine again. But now it's just as bad as before. Can I come for another aspiration?"

Subsequent clinical course

The knee joint was aspirated on three further occasions in the space of one month. On the third occasion, 10 mg triamcinolone was administered intra-articularly in the hope of stabilising the "activated OA".

Four days later, Mr.Miller telephoned to say that the pain had become unbearable and asked the doctor to pay him a call at home.

Findings

The knee was now very red, swollen, and extremely tender; an exploratory aspiration yielded cloudy yellowish synovial fluid. Since the doctor suspected septic arthritis, he had Mr. Miller admitted at once to the local hospital. Here, a culture was made from the synovial fluid, leading to a diagnosis of infection with *Staphylococcus aureus*.
In response to repeated irrigation of the joint and antibiotic treatment in large doses, the patient's condition gradually improved, but it was one month before he could be discharged from hospital.

Diagnosis: Septic arthritis

Intra-articular injections

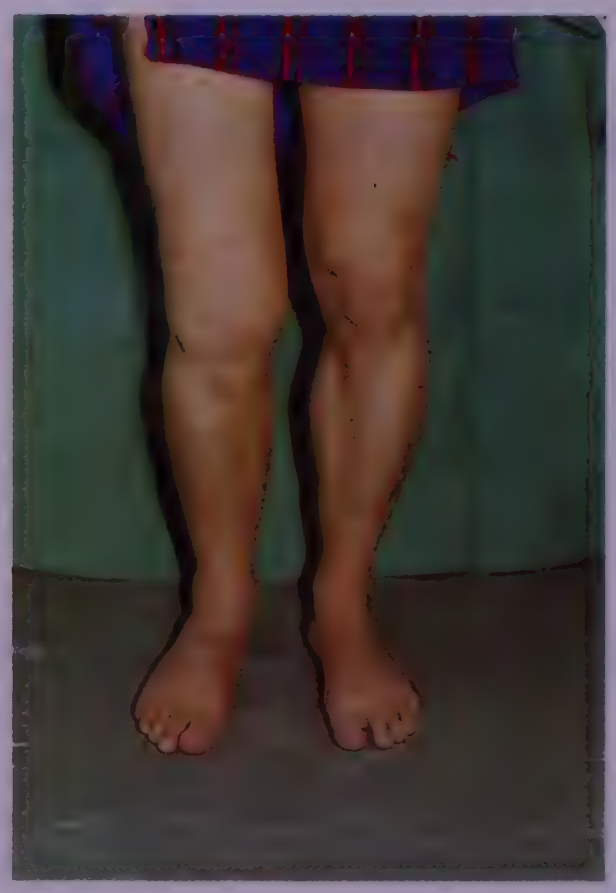
1. Where treatment is administered by an experienced physician, infectious arthritis very seldom occurs following intra-articular injection of steroids.
2. Such injection treatment, however, must be confined to the correct indications
   - Arthritis with an established diagnosis
   - Particularly troublesome involvement of a single joint in patient with rheumatoid arthritis
   - Flare of OA.
3. Not more than three injections per year should be given into any one joint.
4. Clean technique
   - There must be no skin lesions or infections in the neighbourhood of the injection site
   - Degrease the site
   - Disinfect the site (and do not touch it again afterwards)
   - Do not touch the injection needle
   - Employ disposable syringes and needles; do not use multiple-dose ampoules.
5. Possible complications
   - Septic arthritis
   - Microcrystal-induced arthritis
   - Diabetic decompensation.

Case 2:

Mr. Brown, a 53-year-old teacher of florid complexion and somewhat overweight, whom the doctor has known for years, hobbles into the consulting room with an expression of pain on his face.

"That's a bad limp you've got, Mr.Brown."

"Yes, my right knee is frightfully painful, and it's been getting more and more swollen since yesterday. Last night in bed I hardly knew what position to rest it in. And I've been perspiring a lot too."



"Have you ever had such pain in the knee before?"

"Yes, about six months ago; and about two months ago I also had severe pain in the same knee, but then I took some pain-killing tablets from the medicine cabinet at home and they did the trick at once."

Examination

The joint contours of the right knee are barely visible, the skin is hot, and there is a taut fluctuant swelling with a patellar tap sign. Full extension of the knee is prevented by the onset of pain in the popliteal fossa.

"Mr. Brown, have you recently had a heavy meal, perhaps with a fair amount of alcohol?"

"Yes, a couple of days ago we had a family celebration."

"Well, to find out whether your feast may have brought on an attack of gout, I'll now put a needle into the joint and take off as much fluid from it as possible. That'll lessen the pain at once, and then – with the help of the microscope – we may perhaps be able to diagnose the trouble right away."

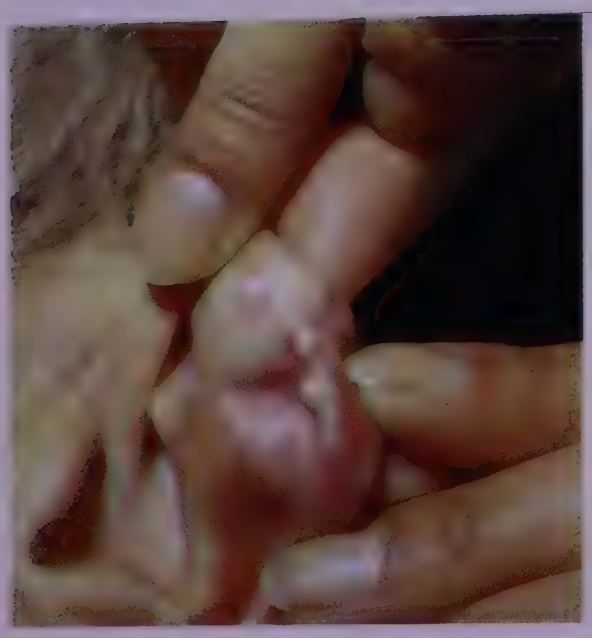
The joint is aspirated and 35 ml of slightly opaque synovial fluid removed. The drop drips from the needle without forming a thread. Urate crystals are visible under the microscope.

Additional subsequent findings

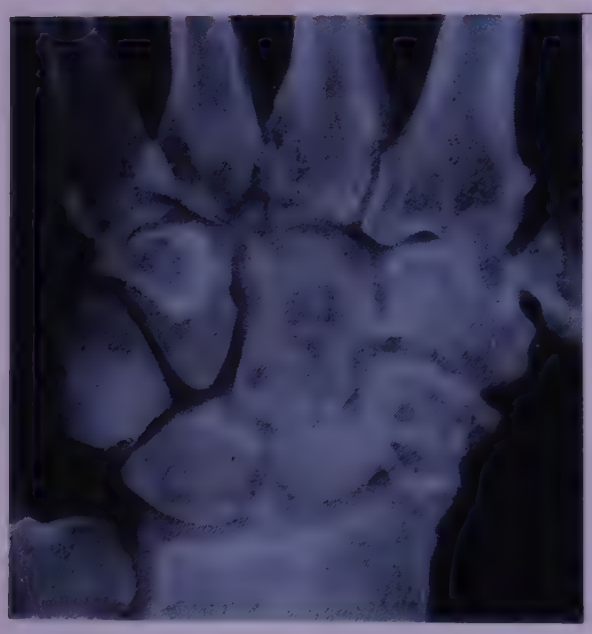
ESR 22 mm/h, haemoglobin 142 g/litre, uric acid 535 µmol/litre.
Synovial fluid culture: negative.

"Yes, it's an attack of gout all right. I'll prescribe some tablets for it. Rest the knee at home in the raised position and keep it cool. Here's a little pamphlet that'll tell you all about gout: read it through carefully and, at the next consultation, we can deal with any points you may still be in doubt about."

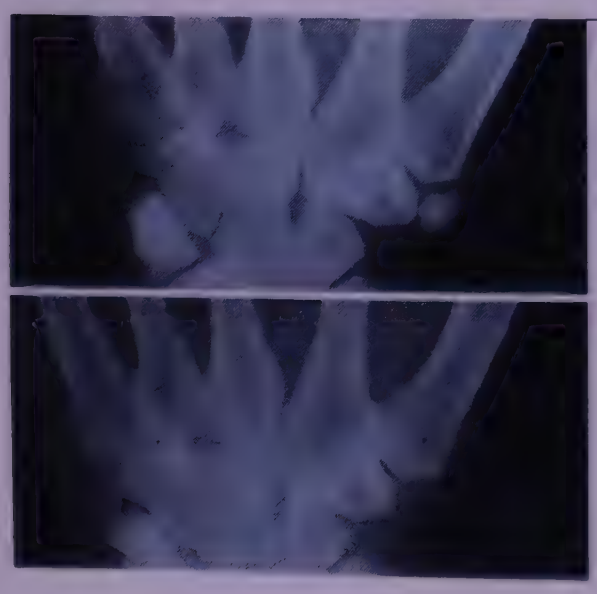
Diagnosis: Crystal-induced arthritis (acute attack of gout)



Tophi may be very unobtrusive, and one has to look carefully for them!



Chondrocalcinosis: typical calcification of the triangular ligament in the wrist.



Hydroxyapatite deposit. The lower picture (taken one month later after an episode of acute arthritis of the wrist) shows that the deposit has now disappeared.

Crystal-induced arthritis

Gout

Diagnosis based on

- Presence of urate crystals in the synovial fluid or in a tophus
- Hyperuricaemia.

Gout occurs in men as from the age of 30; in women it is not encountered until after the menopause (and then it often assumes an atypical form in which several joints may be affected even in the incipient stages of the disease). In elderly patients it is frequently precipitated by thiazide diuretics.

Chondrocalcinosis

Diagnosis based on

- Presence of calcium pyrophosphate crystals in the synovial fluid
- Typical calcium deposits in cartilage.

Occurs chiefly in elderly people.

Hydroxyapatite rheumatism

Diagnosis based on

- X-ray evidence of multiple periarticular calcium deposits, which may disappear again after an attack of arthritis.

Is relatively uncommon.

Treatment for gout

Acute attack

Anti-inflammatory medication in high doses for a few days.

Local steroid injection, provided the possibility of septic arthritis can be excluded.

If more than 2–3 attacks are occurring per year

Reduce the uric acid concentration in the body

1. Instruct the patient to drink plenty of water
2. Advise him to avoid foods rich in purine: sausage meat, offal, etc.
3. Uricosuric medication or
4. The xanthine-oxidase inhibitor:

Allopurinol: 100–300 mg daily.

Note that any sudden change in the uric acid concentration may precipitate an attack of gout: therefore adopt an incremental dosage schedule; ensure that a high volume or urine is maintained, and administer prophylactic therapy for 2–3 months with:

Colchicine: 1 mg daily.

Asymptomatic hyperuricaemia (≦ 10 ng/100 ml; ≦ 600 µmol/litre) is harmless and should not be treated with drugs (possibility of side effects, costs).
Since treatment is long-term, it is indicated only in cases of genuine gout.

Case 3:

Sonia, a 12-year-old girl, holds out her left hand to greet the doctor, while keeping her right arm folded across her body.

"So you're shaking hands with the left hand now!"

"Yes, Doctor, I dare not use the right arm, because my elbow has been so painful since yesterday."

"Let's have a look at it."

Examination

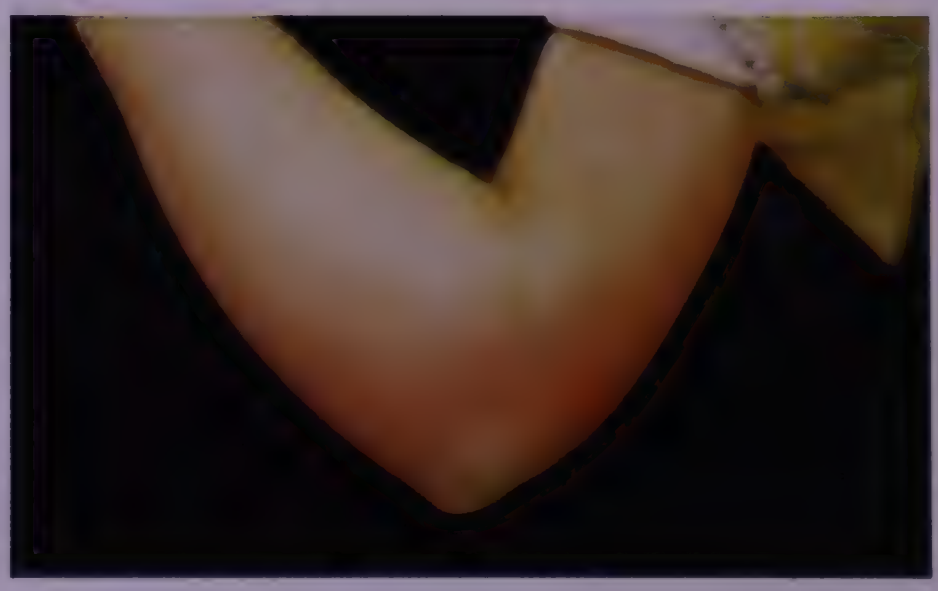
Over the swollen right elbow, which the patient holds in a guarded position, the skin is hot and red; the whole elbow is extremely tender, and a bulging effusion is palpable, particularly in the neighbourhood of the ulnar epicondyle.

"Have you got pain in any other joints?"

"No – except that this morning it felt as if the left elbow and both of my hands were a bit swollen, but they're all right again now."

"Have you been feeling feverish lately?"

"Yes, about ten days ago, Mummy kept me home for a few days because I was running a temperature and because I also had a funny sort of skin rash."



"What did it look like?"

"I had little red spots, like confetti under the skin."

"Were you the only one in the family with this rash?"

"No, both my brothers had the same thing two weeks before I did."

"Didn't they complain of pain in the joints?"

"No."

Skin rash in a case of rubella.

Treatment

Application of a cold compress and prescription of analgesic tablets. Four days later, Sonia visits the doctor again:

"Well, how's your elbow now, Sonia?"

"Since yesterday I've had no more pain; this morning the elbow still felt a bit stiff, but now the stiffness too has almost disappeared."

"There's no doubt that what you've had is German measles (the doctor was certain of this, because there had been an epidemic of German measles in the village). When children and teenagers catch the disease, it's by no means unusual for them to develop joint inflammation about a week after the onset of the illness. But this inflammation is quite harmless and completely disappears again. Beware: measles during early pregnancy are dangerous for the developing baby. Girls should be vaccinated before the age of 12–13 years.

Diagnosis: Arthritis due to virus infection

Arthritis due to bacterial infection

In septic forms of arthritis, the pathogen is detectable within the joint. Treatment consists in administration of specific antibiotics selected on the basis of cultures and the antibiogram. The affected joint should be rested and immobilised. Surgical drainage is seldom necessary.

Examples
Staphylococcal arthritis
Tuberculous arthritis
Gonococcal arthritis.

In cases of reactive arthritis, joint involvement becomes apparent some time after the infection.
Although antibiotic treatment does not influence the clinical course of the arthritis, it may serve to prevent recurrences.

Examples
Post-streptococcal arthritis
Reiter's syndrome.

Arthritis due to virus infection

Encountered in Europe are:

Arthritis due to rubella

Chiefly in postpuberal patients

Occurs more frequently in females

Also liable to occur after vaccination

Clears spontaneously

Mumps arthritis

Occurs more frequently in males

Clears spontaneously

In both these forms of arthritis the virus can be detected in the synovial fluid.

Arthritis due to hepatitis B

Usually starts a few days before hepatitis becomes clinically manifest

Often associated with urticaria and fever

Clears spontaneously following the onset of jaundice.

(The viral antigens and the antibodies form immune complexes like those occurring in serum sickness.)

A number of other viruses are also known to induce arthritis (e.g. parvovirus).

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Rheumatology in everyday practice

Osteoporosis

P. Geusens and J. Dequeker

Vol.4

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1. Skoutakis, V.A.; Carter, C.A.; Mickle, T.R.; Smith, V.H. et al.; Review of diclofenac and evaluation of its place in therapy as a nonsteroidal antiinflammatory agent. Durg Intell and Clinical Pharmacy. 22:850-859, 1988.

Osteoporosis

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Case 1

Mrs. W., a small and slender woman of 54 years, complains of acute back pain after lifting a chair.

"Yesterday, I felt a terrible pain in my back, which kept me awake all night. I feel pains when I stand still and they increase when I take a deep breath."

"What were you doing when the pain occurred, and where do you feel the pains?"

"I was in the middle of cleaning and was just lifting a chair when I felt this pain go right through my rib cage."

Clinical examination

The patient was small (154 cm) and slender (47 kg), having a thin, transparent skin. Armspan was 156 cm. She had a dorsal hyperkyphosis with severe pain on movement and local palpation.

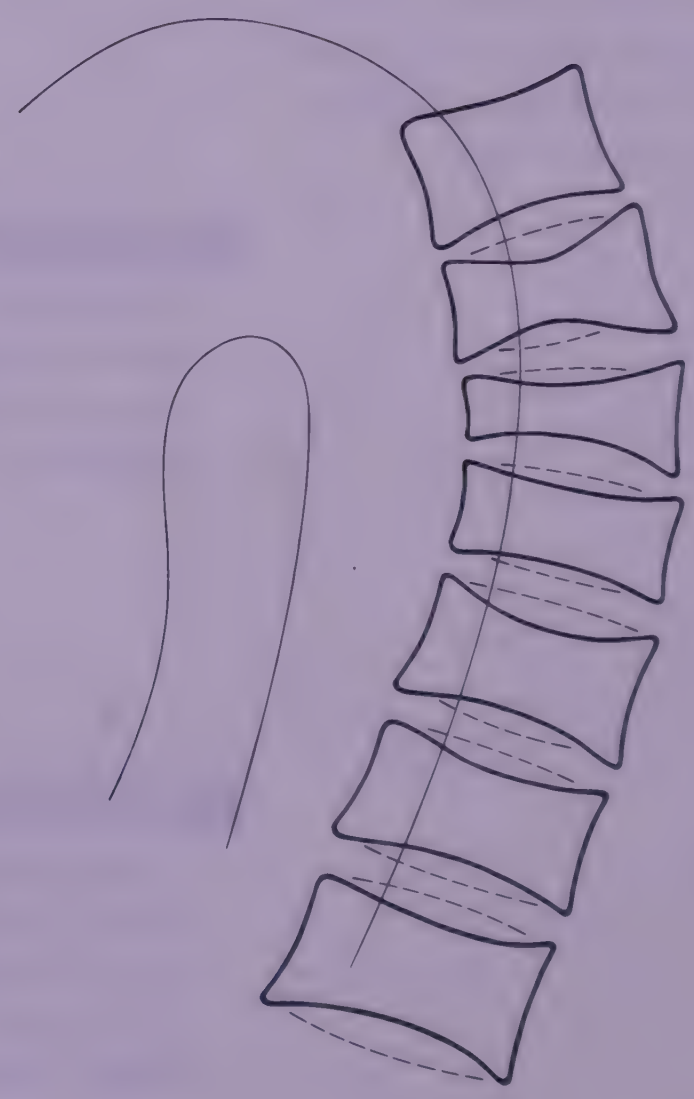
Medical history

Total radical hysterectomy at 35 years because of recurrent menorrhagia. The patient was not given any hormone replacement therapy afterwards because she was anxious about taking "hormones". She has one child. She smokes 10 cigarettes a day. There is no alcohol abuse.

X-rays

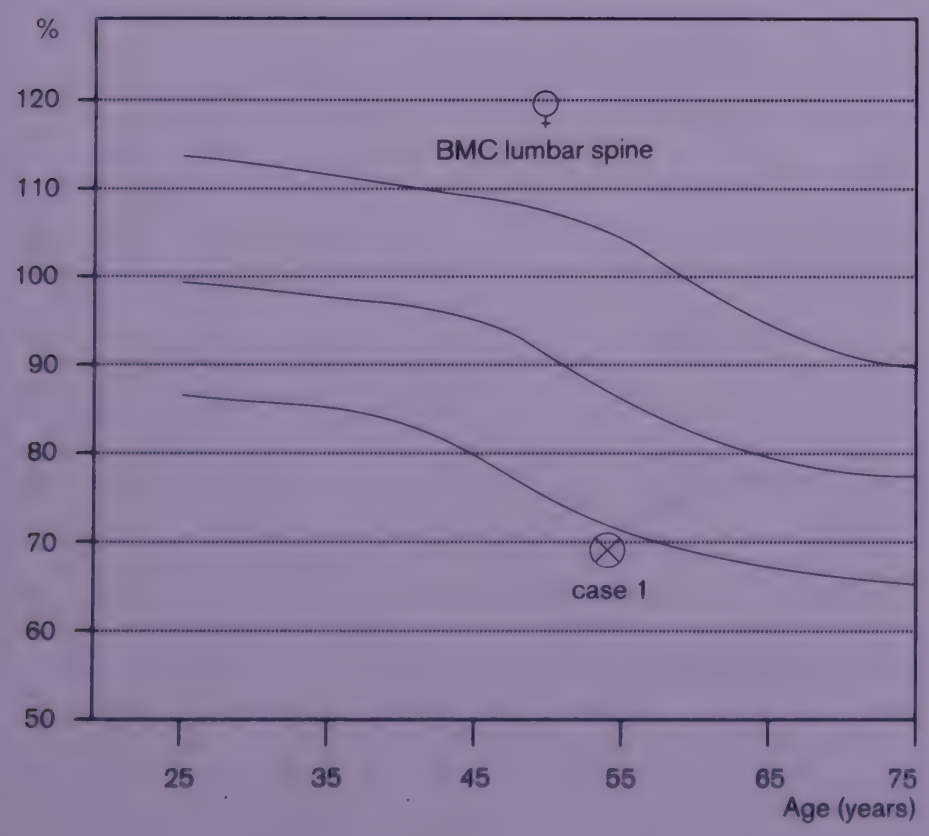
Vertebral collapses with anterior wedging of T5, T6, T7, and L2.

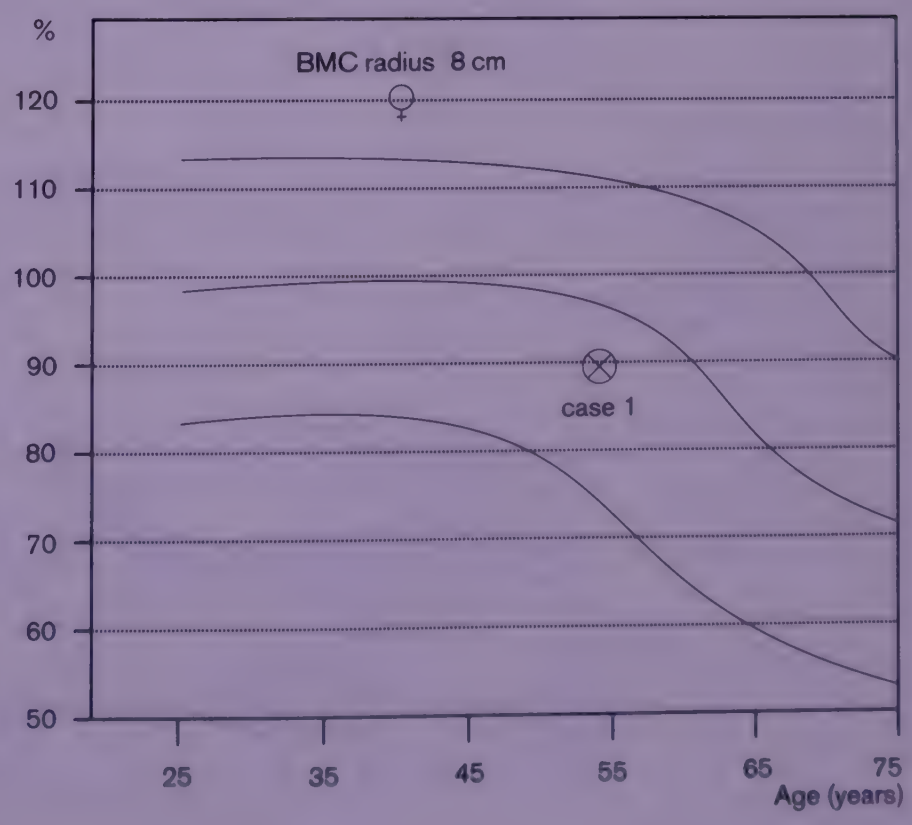
Fig. 1. Radiograph of the thoracic spinal column of Case 1 (osteoporosis after early oophorectomy) with anterior wedging of T5 and T6 (reduction of the anterior height), partial collapse of T7 (reduction of anterior and posterior height), and surface fracture of T5.



Bone mineral content of the radius	
	Low normal values.
Bone mineral content of the lumbar spine	
	Very low bone density, far below the 10th percentile of young, normal, and age-matched controls.

Fig. 2. Age-related change in bone mass of the lumbar spine and the radius (mean ± two standard deviations). The values of Case 1 with osteoporosis after early oophorectomy are indicated.





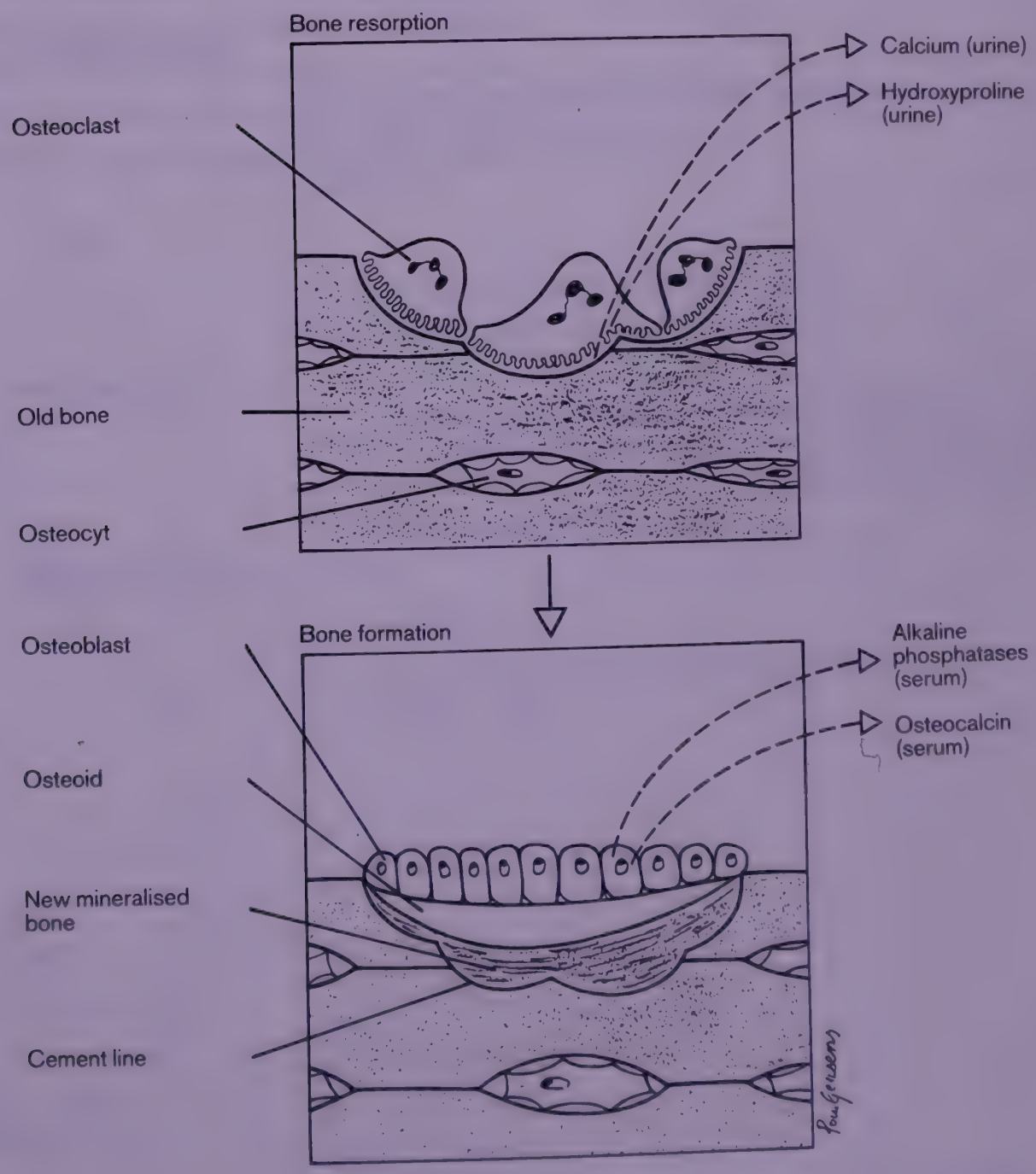
Laboratory investigations

Blood

Normal serum calcium, phosphate, alkaline phosphatase, and osteocalcin. Normal protein electrophoresis.

Urine		Patient	Normal values
2-hour	calcium/creatinine (mg/mg)	0.18	<0.15
	hydroxyproline/creatinine (mg/mg)	0.055	<0.045
24-hour	calcium (mg)	150	100–250
	hydroxyproline (mg)	45	<50

Fig. 3. Bone resorption can be evaluated by determining the urinary calcium / creatinine and hydroxyproline / creatinine ratios in fasting morning urine. Bone formation can be estimated from the serum concentrations of alkaline phosphatase and osteocalcin which result from osteoblast activity.



Diagnosis

Osteoporosis with vertebral crush fractures and pronounced trabecular bone loss as a result of bilateral oophorectomy at a young age without any subsequent estrogen replacement therapy. Several risk factors are known for the development of osteoporosis. The most well-known is early menopause (either spontaneous or following bilateral oophorectomy). Soon after menopause, bone loss is accelerated, especially that of trabecular bone; later on in life, however, it is cortical bone loss which is dominant. Two types of osteoporosis have been defined, as shown in Table 2. Postmenopausal osteoporosis is characterised by fractures occurring at skeletal sites with predominantly trabecular bone (distal radius and vertebrae), while femoral neck fractures occur in senile osteoporosis when trabecular and cortical bone loss has occurred.

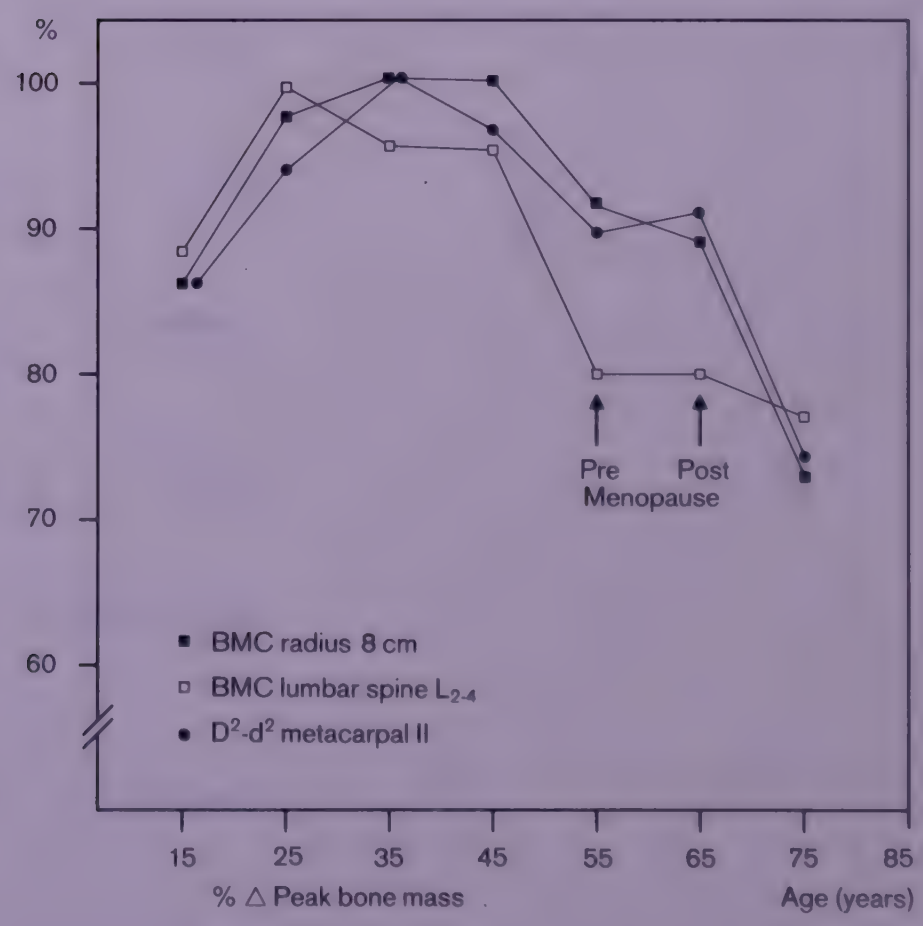


Fig. 4. Age-related changes in peak bone mass of the radius (■), lumbar spine (□), and metacarpals (●) among normal women.

Table 1: Risk factors for osteoporosis	
• Advanced age	
• Genetics	
	women > men white > black familial history of osteoporosis absence of signs of polyarthritis
• Habitus	
	small stature slender and thin skin low weight
• Hormones	
	premature menopause (either spontaneous or as a result of oophorectomy) nullipara
• Diet	
	inadequate calcium intake in youth, after menopause high protein intake
• Lifestyle	
	cigarette smoking alcohol abuse minimal physical exercise
• Intercurrent illness	
	gastrectomy hyperparathyroidism, hyperthyroidism corticosteroids

Table 2: Types of involutional osteoporosis

	Postmenopausal	Senile
Age	51–70	>70
Bone loss	trabecular	trabecular and cortical
Sites	vertebrae, distal radius	vertebrae, hip
Main causes	factors related to menopause	factors related to ageing

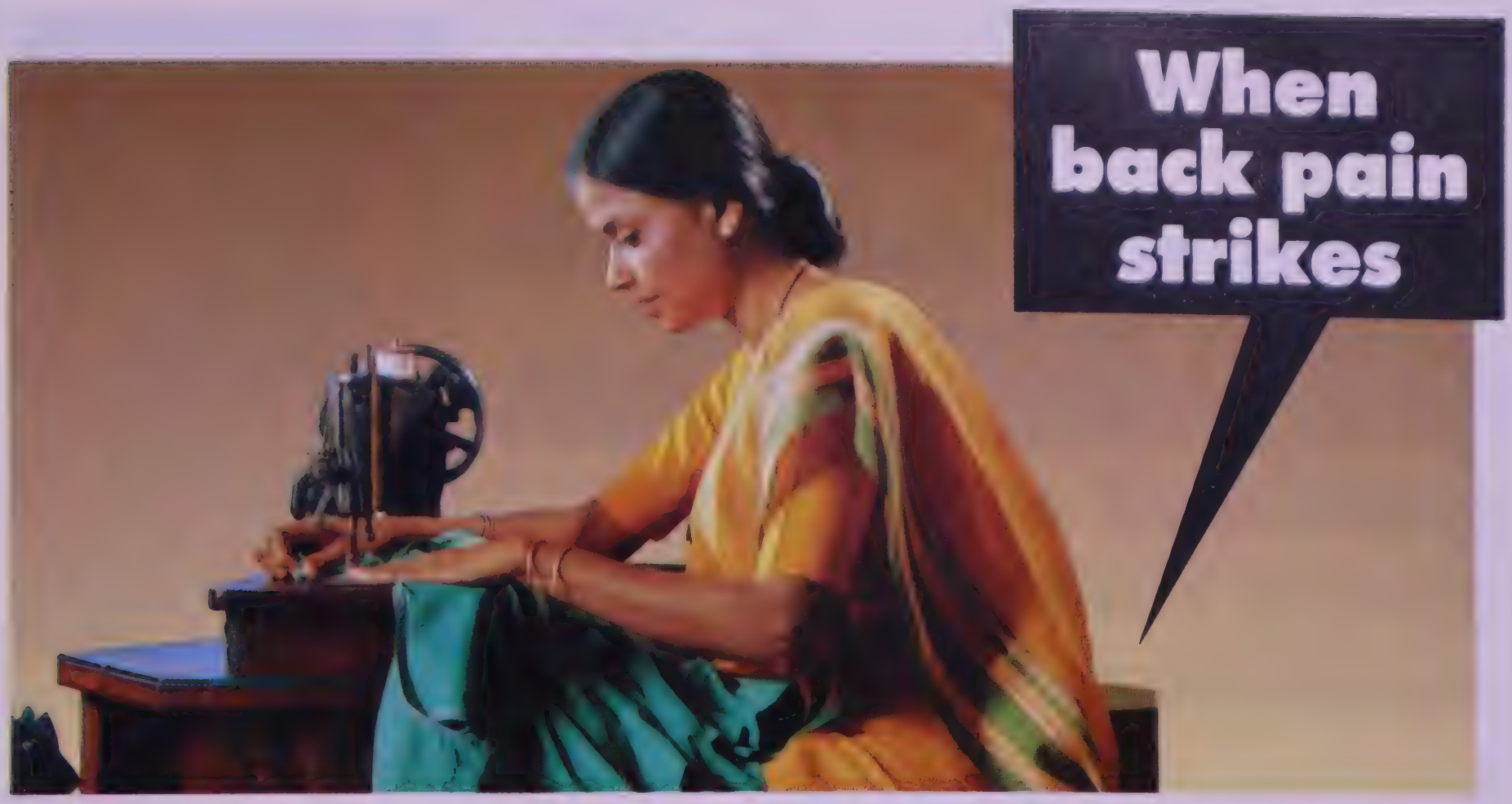
Note: The female:male ratio of involutional osteoporosis narrows from 6:2 before the age of 70 to 2:1 over the age of 70.

Subsequent clinical course

After a period of bed rest and treatment with analgesics, the patient could not be motivated to receive hormone replacement therapy with estrogens and progestogens. Her treatment regimen consisted of oral calcium and exercise. During the next few years she suffered several new fractures, a pubis fracture at 56 and a Colles fracture at 58.

Comments

In this case, preventive therapy could have been of benefit, but should have been started immediately after bilateral oophorectomy. Estrogen replacement therapy stops bone loss, but cannot restore the lost bone once this has occurred following menopause or oophorectomy.



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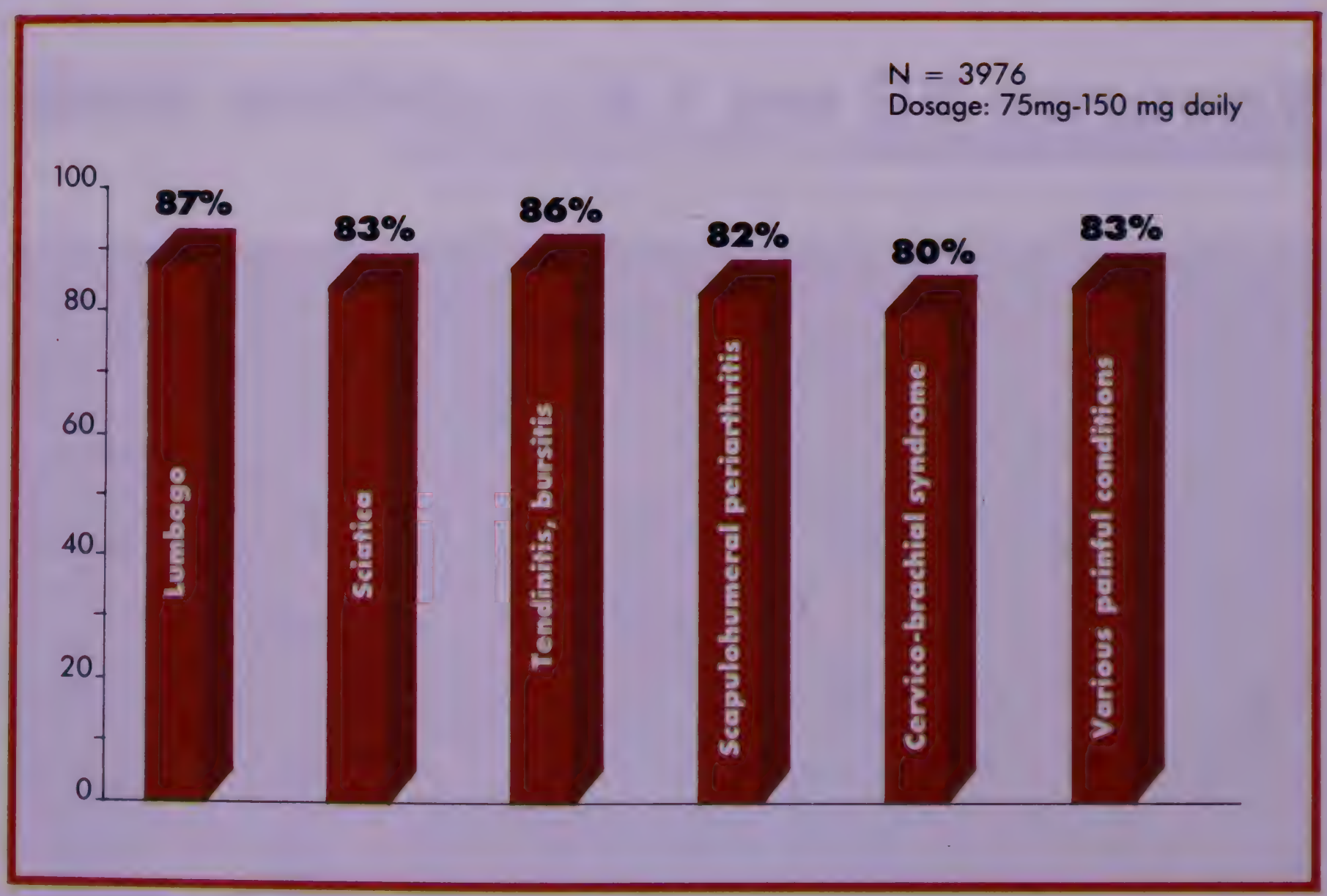


Excellent to good results in percentage of patients, reported according to type of pains.[1]

1. Adapted from Rivet, J.P. Richard, A: Voltaren an anti-inflammatory drug in rheumatology: Clinical assessment of 10,352, patients by 2623 General Practitioners in France. Gaz Med. Fr. 84, 35471, 1977.

Case 2

At 50 years of age Mrs. G. underwent a hysterectomy without oophorectomy for fibromatosis. Two months after the operation she consulted a rheumatologist because of severe right ankle and knee pain, with diffuse swelling of the knee, the lower leg, and the foot. The patient was treated with analgesics but the pain and swelling persisted. After two years she reported progressive severe low-back pain without a history of trauma.

Clinical examination

The patient had a prominent dorsal hyperkyphosis, severe pain on examination of the back, a swollen right lower leg, pain on moving the knee and ankle, and local pitting oedema. She was 145 cm tall, weighed 58 kg, and had an armspan of 155 cm.

Laboratory investigations

Blood tests for calcium and phosphorus metabolism, as well as for signs of inflammation were negative. The two-hour fasting morning urine showed increases in the calcium / creatinine ratio and the hydroxyproline / creatinine ratio.

X-rays

Vertebral collapses of T6, T7, T10, T11, L2, and L4. Patchy periarticular osteoporosis of the right ankle.

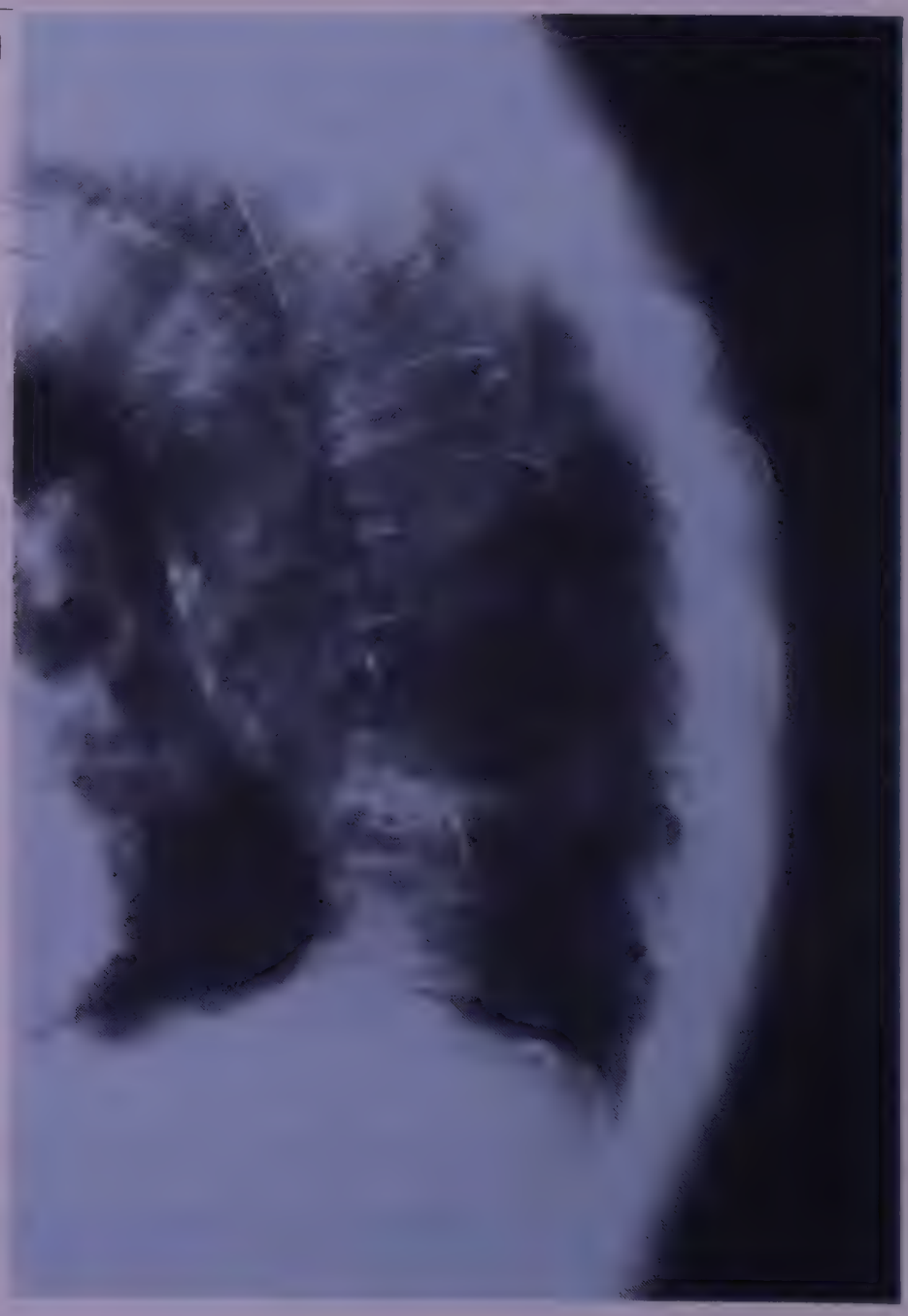
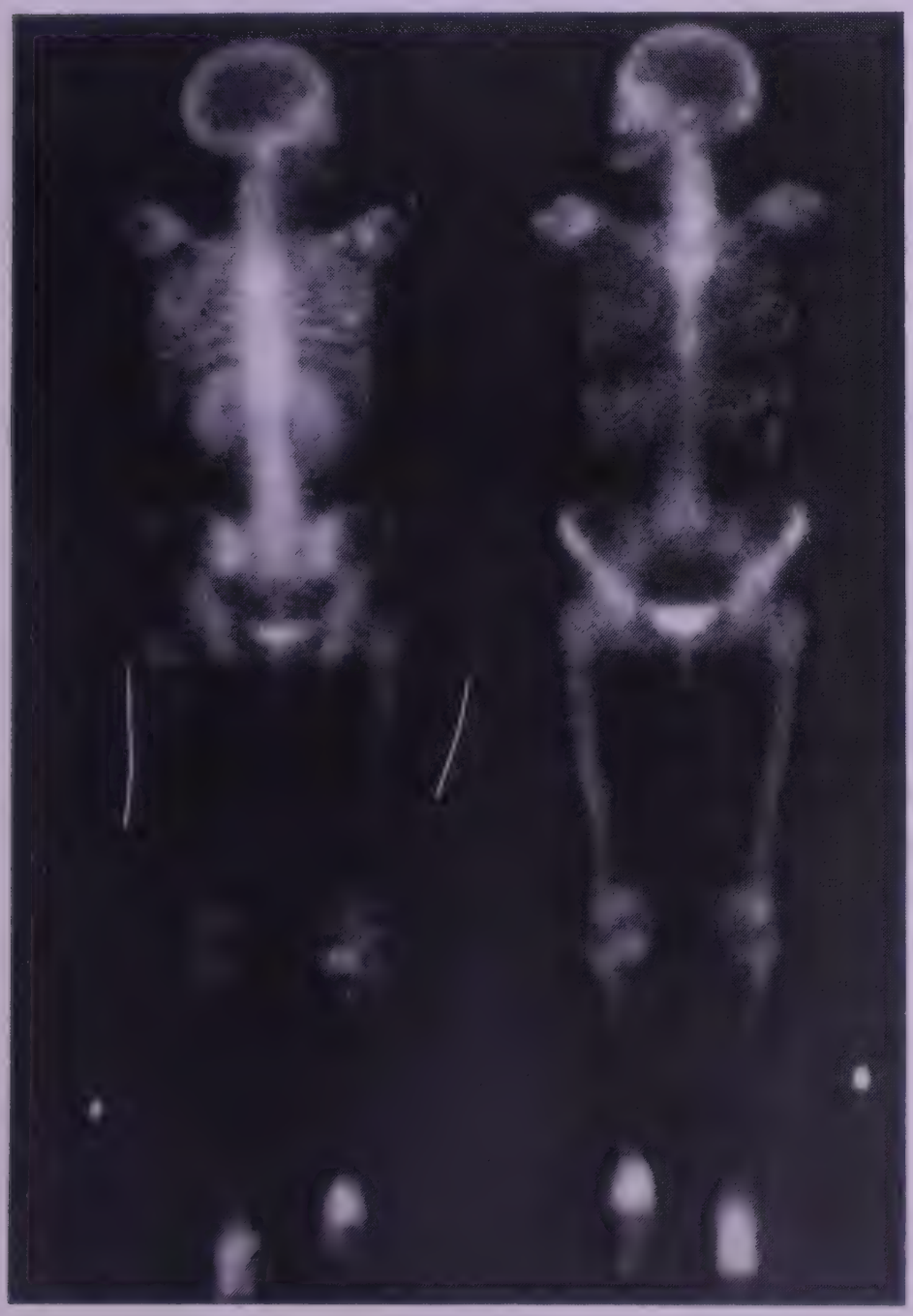


Fig. 5. Thoracic spinal column of Case 2. Vertebral collapse and wedging of several vertebrae.

Technetium-99m bisphosphonate bone scan
Increased uptake in several thoracic and lumbar vertebrae and in the left foot.

Fig. 6 and 7. ^{99m}Tc-bisphosphonate bone scan of Case 2 with algoneurodystrophy and osteoporosis. The scan shows increased uptake at several thoracic and lumbar vertebrae, as well as in the left foot and right ankle.



Diagnosis

Algoneurodystrophy (reflex sympathetic dystrophy), followed by subsequent multiple vertebral crush fractures.

Subsequent clinical course

The patient was treated with calcitonin subcutaneously which resulted in diminution of pain, decrease in calcium and hydroxyproline excretion, and inhibition of further bone loss.

Comments

Algoneurodystrophy of the spine has been reported sporadically in the literature, and was in most cases associated with peripheral reflex sympathetic dystrophy. Post-traumatic vertebral collapse has also been described after minimal trauma of the vertebral column. These conditions are characterised by a marked increase in bone turnover, and treatment with calcitonin inhibits further bone loss. Calcitonin also has analgesic properties, which are believed to be the result of an effect on the central nervous system.

Case 3

Mr. S. presented with a long history of multiple fractures over the last five years, from the age of 54. He had had several vertebral crush fractures without trauma, and multiple peripheral fractures after a fall, localised at the femoral neck, the clavicula, the distal femur, the humerus, and the radius. He is a heavy smoker and drinker with chronic bronchitis, aggravated by hyperkyphosis of the dorsal spine and rib fractures. He has never been treated with corticosteroids for his pulmonary problems.

Clinical examination

Severely invalid patient who walks on crutches. Severe hyperkyphosis with extreme dyspnoea. Muscular atrophy of arms and legs.

Radiology

Multiple fractures of the axial and appendicular skeleton. Osteosynthesis of bilateral femoral neck fractures. An X-ray of the hands shows a Colles fracture of the right radius and generalised thinning of the cortical bones.

Fig. 8. Radiograph of the lumbar spine of Case 3 with multiple codfish deformities of the vertebrae.

Fig. 9. Radiograph of the pelvis and hip of Case 3, with bilateral femoral neck fracture treated with osteosynthesis.

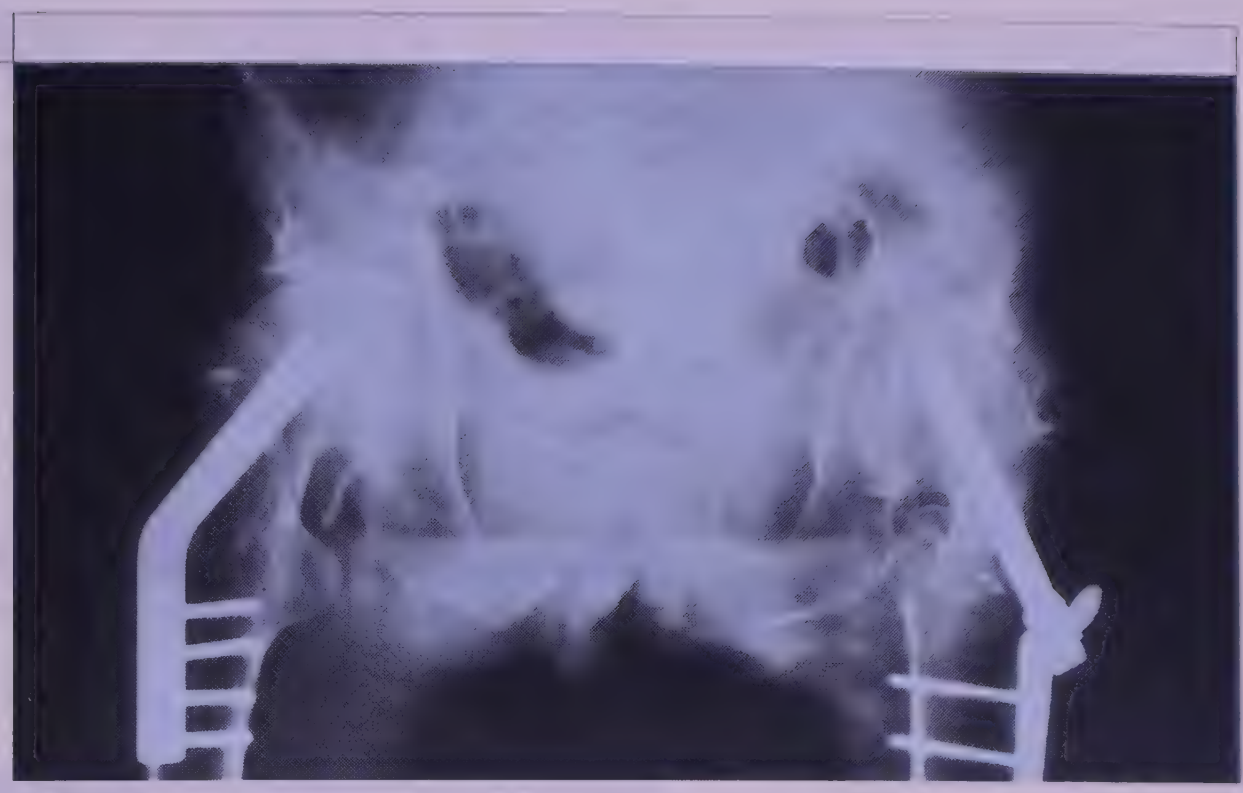
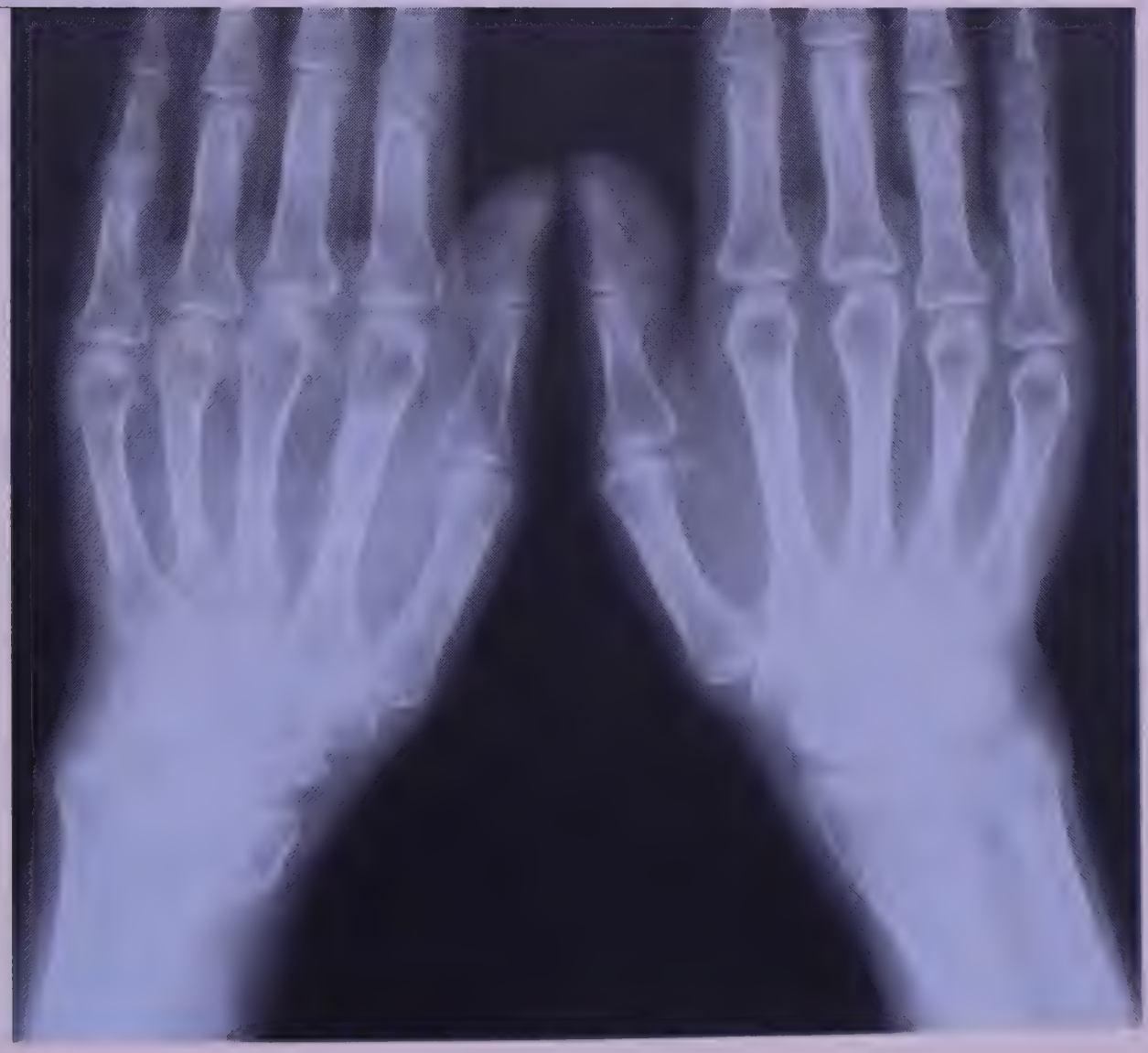


Fig. 10. Radiograph of the hands with left Colles fracture and general thinning of the cortical bone.



Laboratory investigations

Calcium and phosphorus metabolism parameters were normal in serum and urine. Values of 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D in serum were normal, as was serum parathyroid hormone. No hypercalciuria was observed.

Diagnosis

Idiopathic osteoporosis of a man with a history of nicotine and alcohol abuse.

Treatment

Anabolic steroids produced an increase in the bone mineral content of the radius at one and two years.
The patient was later treated with sodium fluoride, vitamin D, and calcium; this regimen led to an increase in lumbar spine bone mineral content with simultaneous reduction in the bone mineral content of the radius.

Subsequent clinical course

The patient died as a result of cardiovascular problems and respiratory insufficiency secondary to chronic bronchitis and to a diminished thoracic volume and restricted expansion caused by the vertebral crush fractures and multiple rib fractures.

Comments

Osteoporosis in men has a variety of causes. Alcohol and nicotine abuse increases the risk for fractures. In men, osteoporosis is often associated with hypercalciuria, which was not so in this case at the time of fractures. Patients with hypercalciuria can be treated with thiazides.

Severe osteoporosis with vertebral and rib fractures can lead to pulmonary and cardiovascular problems. In such cases, anabolic steroids represent a useful therapeutic approach, augmenting cortical and trabecular bone and increasing muscle mass.

Table 3: Causes of osteoporosis in men

- Hypogonadism
- Gastrectomy
- Hypercortisonism (endogenous or exogenous)
- Hypercalciuria
- Alcohol and nicotine abuse
- Low calcium intake

Case 4

At the age of 36, Mrs. Ch. was treated for severe anorexia nervosa. She weighed 29 kg and had multiple vertebral crush fractures. She was 143 cm tall, having lost 15 cm of her normal height. Her armspan was 159 cm. She smoked 20 cigarettes a day. She suffered a fracture of the pubis when she fell off a bicycle. She had a very low bone mass of the lumbar spine, with normal bone mineral content of the radius. She was given cyclic hormone replacement therapy with estrogens and progestogens, after which no further bone loss occurred in the lumbar spine or the radius.

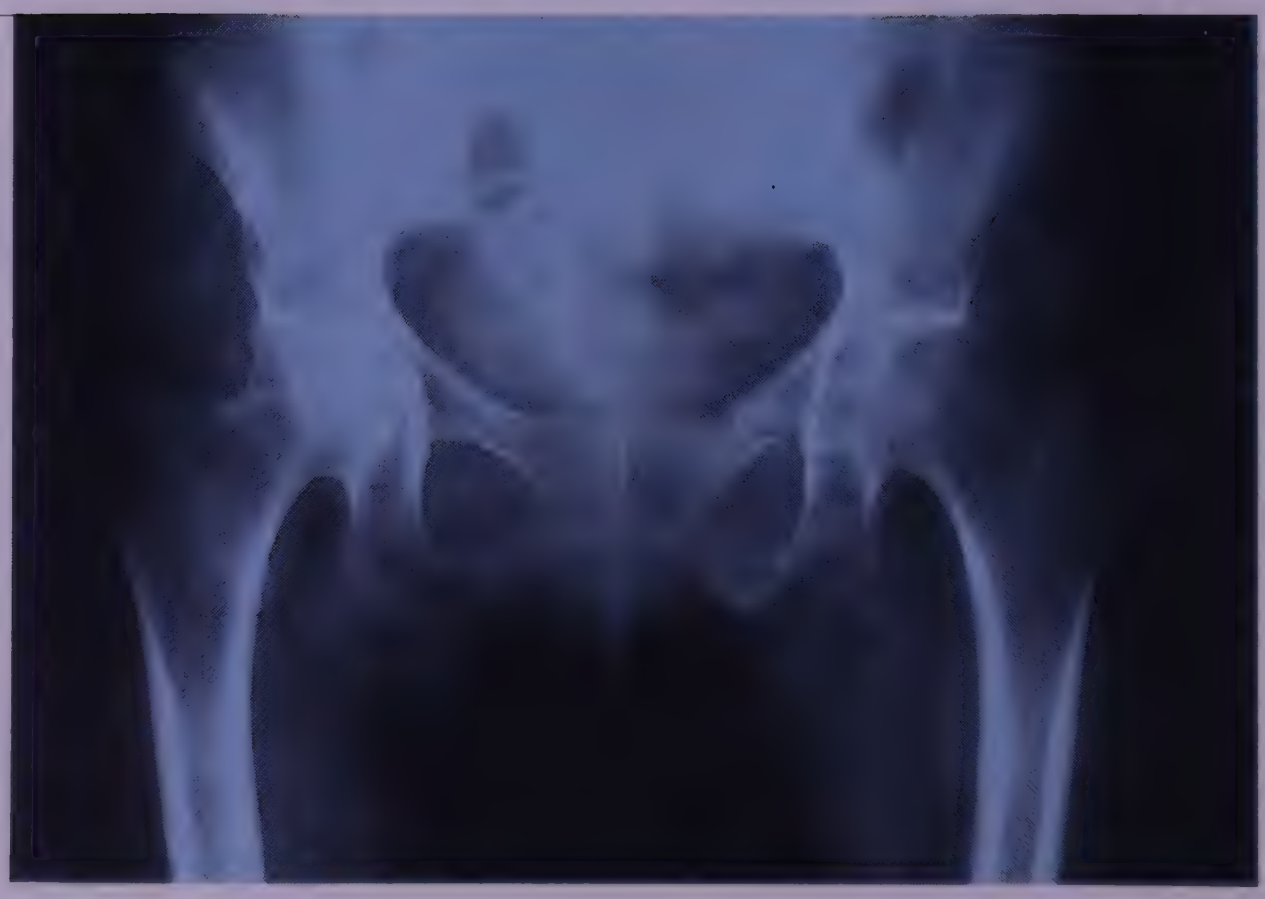


Fig. 11. Radiograph of the pubis of Case 4 with anorexia nervosa, displaying a fracture of the pubis.

Comments

Functional hypogonadism with secondary amenorrhoea occurs in anorexia nervosa and also in female distance runners, resulting in the kind of bone loss seen after menopause. At the age of 36, the patient already had a bone mass within the zone of fracture, which is two standard deviations below the peak bone mass of young healthy women, putting her at increased risk of fracture. Subcutaneous fat plays an important part in the prevention of osteoporosis. Patients of low body weight have less subcutaneous fat. Obese patients, on the other hand, have rather a lot, and it is here in this fatty tissue that androstenedione, a product of the adrenals, is converted to estrone after menopause. This results in higher circulating estrogen levels, which protects against bone loss. Indeed, patients with obesity and/or osteoarthritis have been found to have a higher bone mass. An important contributing factor in this case was also the patient's smoking habits. Cigarette smoking exerts an anti-estrogenic effect by increasing the breakdown of estradiol to less active metabolites.

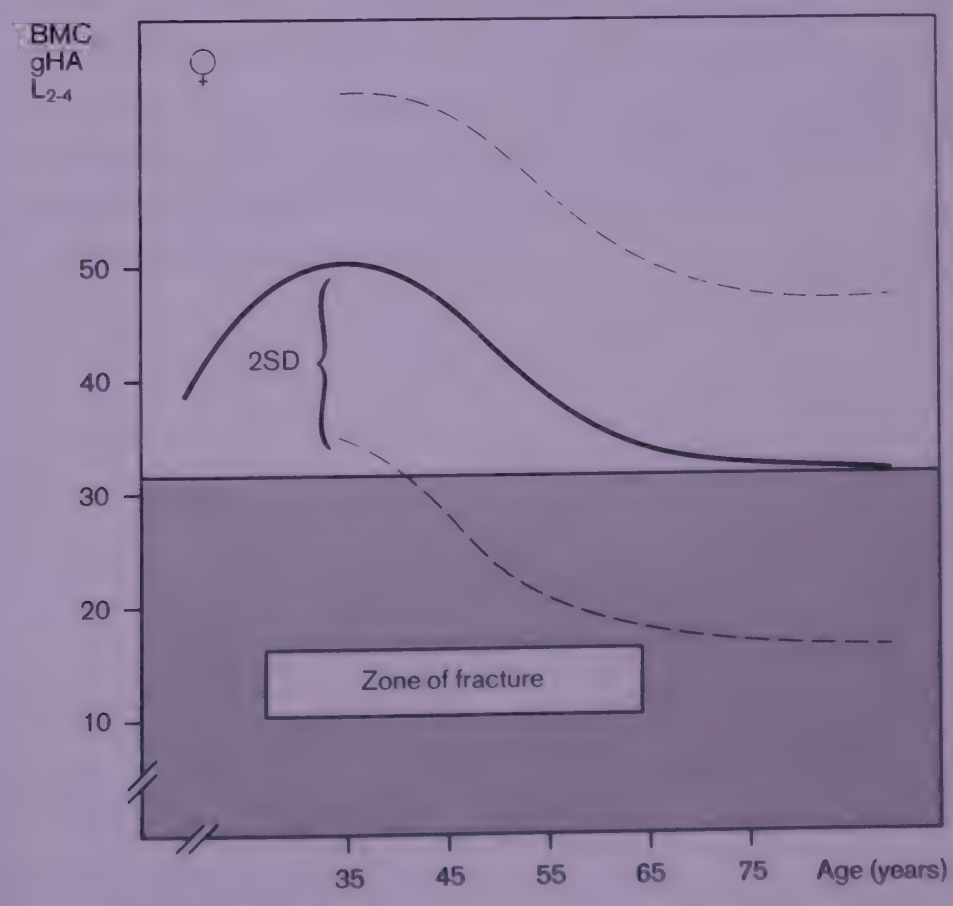


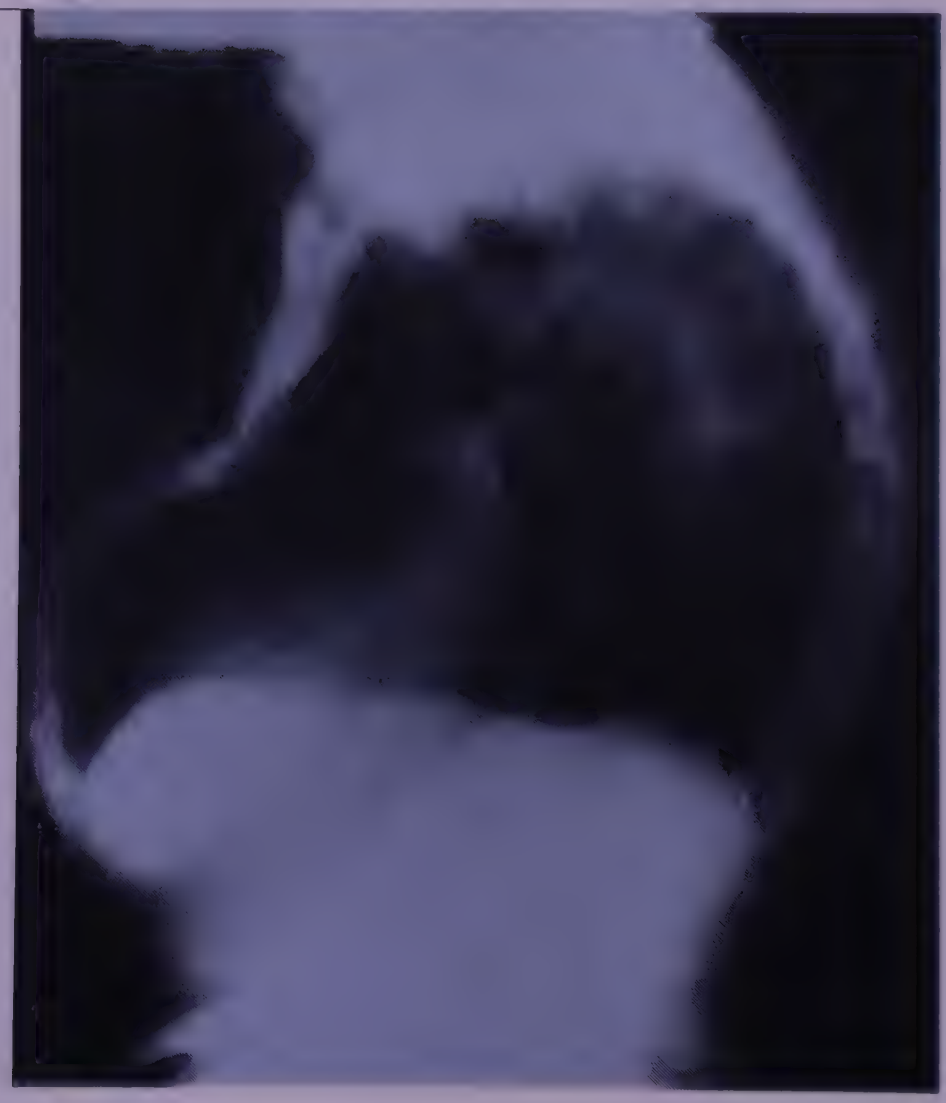
Fig. 12. Changes in the bone mineral content BMC of the lumbar spine (in grams of hydroxyapatite) as a function of age. The theoretical zone of fracture is reached when the BMC is approximately two standard deviations below the mean peak bone mass at a young age. At 75 years of age, 50% of the normal population is within this fracture zone.

Case 5

[redacted]

Mrs. C., aged 65.
This patient has suffered from asthma-like bronchitis from the age of 16 and had been treated for more than 20 years with corticosteroids.
She consulted the doctor because of vertebral crush fractures and sternum and rib fractures from the age of 57.
Measurements of bone mineral content revealed a decreased cortical and trabecular bone mass.
She was treated with calcium supplements and vitamin D.

Fig. 13. Corticoid-induced osteoporosis (Case 5) in chronic bronchitis with emphysema and dorsal hyperkyphosis with multiple vertebral crush fractures and fractures of the sternum.



Diagnosis

Secondary osteoporosis as a result of long-term high-dose corticosteroid therapy.

Comments

In a patient with fractures, causes of secondary osteoporosis have to be excluded. The most common causes are early oophorectomy (in women), hypogonadism (in men and women), subtotal gastrectomy, chronic obstructive pulmonary disease, immobilisation, pharmacological doses of glucocorticoid and thyroid hormones, multiple myeloma, and disseminated carcinoma. Glucocorticoid treatment suppresses osteoblast activity, diminishes the calcium absorption of the gut, increases calciuria, and lowers serum calcium, which in turn leads to secondary hyperparathyroidism. This secondary hyperparathyroidism stimulates the osteoclasts into stepping up bone resorption. Glucocorticoids also suppress the pituitary feedback of sex hormone production. The net result is a negative balance between increased bone resorption by osteoclasts and decreased bone formation by osteoblasts.

Table 4: Frequent causes of secondary osteoporosis

• Endocrine diseases	
	Hypogonadism, amenorrhoea
	Corticosteroids (endogenous or exogenous)
	Hyperthyroidism
	Hyperparathyroidism
• Gastrointestinal diseases	
	Gastrectomy
	Malabsorption
	Liver diseases
	Anorexia nervosa
• Bone and bone marrow disorders	
	Multiple myeloma
	Osteolytic metastases
• Other	
	Osteogenesis imperfecta
	Immobilisation
	Chronic heparin administration

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Pain in the arm

J.K. van der Korst

Vol.5

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Pain in the arm

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Case 1

Mrs. J., a 53-year-old married housewife with three children:

"Doctor, I've already had pain in my right arm for several months, and it seems to be getting worse. The arm often hurts all day long, especially when the weather is rainy; and sometimes I wake up in the middle of the night because my right hand feels numb and tingling. I'm worried about it. Is it rheumatism or is it just my age?"

"Does your neck hurt?"

"A little."

"Is your neck stiff?"

"No, not really."

"Does your right hand feel cold?"

"Not that I've noticed."

"Can you move your arm and fingers normally?"

"Yes."

"Did you have an accident before all this started?"

After some hesitation:

"No."

"Does it make a difference how you hold your arm?"

"No, but my arm feels so heavy."

You know Mrs. J. to be a robust and hard-working woman, who has quite often required your help for her children, but very seldom for herself. Now she seems really troubled, although – considering the nature of her complaints – you suspect nothing more serious than cervicobrachialgia.

Differential Diagnosis

Pain radiating through the whole arm, and accompanied by paraesthesiae of the hand, might be due to:

1. Cervicobrachialgia
2. Neurovascular compression syndrome
3. Shoulder-hand syndrome
4. Lesion of the cervical nerves or nerve roots
5. Peripheral entrapment neuropathy.

Case history

You first take her history. Here the following questions may prove helpful:

Clinical picture of cervicobrachialgia

Cervicobrachialgia is by far the most common cause of pain radiating through the whole arm, and it is very frequently accompanied by paraesthesiae of the hand. Whereas the pain occurs mostly during the day, the paraesthesiae often set in at night. Cervicobrachialgia occurs especially between the age of 40 and 65 years and is more common in women than in men. Neck pain and stiffness of the neck may be a prominent feature, but sometimes they are negligible.

It now seems even more likely that Mrs. J. is suffering from cervicobrachialgia.

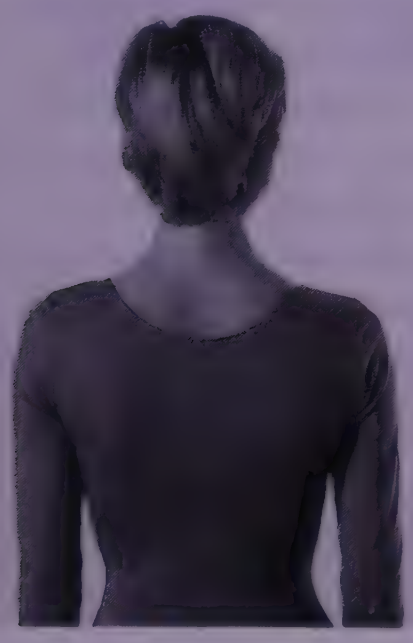
Physical examination

Inspection

You ask the patient to strip to the waist, and you then start looking for the following:

Normal positioning of the neck?	There seems to be an increased lordosis of the cervical spine.
Symmetrical positioning of the shoulders?	Yes.
Muscle atrophy of the shoulders and the arm?	None.

Fig. 1. Symmetrical positioning of the shoulders



Palpation

Is there painful hypertonia of the trapezoid muscles?	Yes, bilaterally.
Can you feel any difference between the right and left radial artery pulse?	No.
Is there a difference between the temperature of the two hands?	No.
Does it hurt the patient when you palpate the lateral and medial humeral epicondyles?	Yes, they are more sensitive on the right than on the left side.

Fig. 2. Examination of the shoulder joints

Passive mobility

Now, with the patient seated, you move her around to test for passive mobility.

Gently bend her neck forwards, backwards, and sideways, and then rotate the neck.	Rotation and flexion of the cervical spine to the right are limited, and full rotation or flexion is somewhat painful.
Examine the shoulder joint (do not forget to fix the scapula!): abduction, anteflexion, elevation, and external rotation.	In uncomplicated cervicobrachialgia there are unlikely to be any serious restrictions of movement in the glenohumeral joint.
See whether the patient can stretch her fingers normally as well as clench her fist.	Yes.

Neurological examination	
Compare the reflexes in both arms.	There should be no difference.
Test both arms and hands in order to gain a rough impression of their sensibility.	There should be no difference.

Fig. 3. Rotation and flexion of the cervical spine

Signs and symptoms of cervicobrachialgia

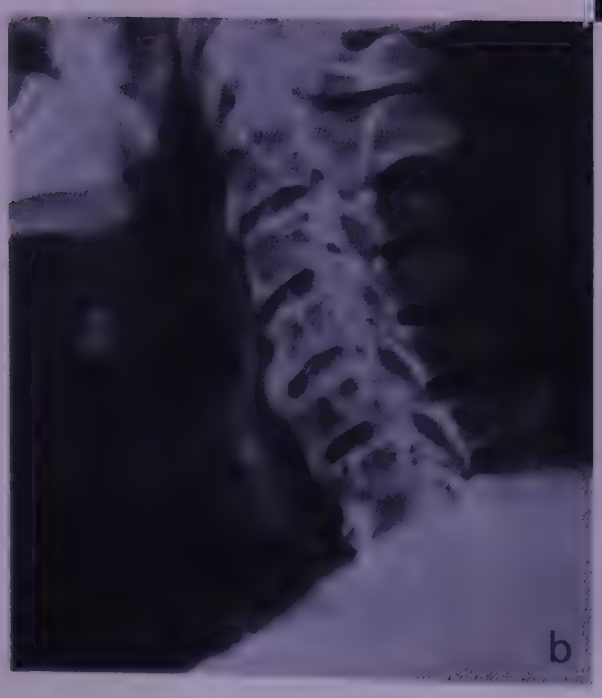
Fig. 4. Painful bony prominences of the arm

In cervicobrachialgia there is a conspicuous lack of objective signs, apart from some restriction of movement in the cervical spine and hypertonia of the trapezoid muscle(s). If there are objective neurological signs, e.g. areas of hypaesthesia, differences in reflex, or loss of muscle strength, the patient should be referred for further neurological examination. In cervicobrachialgia there is usually more or less marked tenderness of bony prominences, such as both epicondyles and the styloid processes of the radius and the ulna.

You now have enough "circumstantial evidence" to warrant a diagnosis of cervicobrachialgia. Should you arrange for laboratory tests to be carried out? Not necessarily, although determination of the erythrocyte sedimentation rate might provide reassurance for the patient or, perhaps even more so, for you yourself. Should you have X-rays made of the cervical spine? Preferably not, as they will be of no help in confirming or denying the diagnosis of cervicobrachialgia.

Note: The patient with the "normal" X-ray (left) has cervicobrachialgia – not so the one with the spondylotic cervical spine.

Radiology in cervicobrachialgia



Although cervicobrachialgia is probably due to degenerative changes in and around the lower cervical disks – at least in the great majority of cases – there is no correlation whatsoever between the severity of radiological signs of spondylosis and spondylarthrosis and the symptoms of cervicobrachialgia.

Fig. 5. a) X-ray of "normal" spine with only minor calcification in the intervertebral space C5–C6;
b) marked spondylotic changes, especially at C4–C5

Treatment

Now you have to consider the problem of treatment. Here, by far the most important thing to do is reassure the patient and convince her that, although it might take quite a long time, her complaints will eventually disappear without any medical help. You should also advise her as to how she can obtain relief from them by adopting a good position when sleeping, as well as by relaxing for one or two short periods during the day, i.e. by lying down on her back with adequate support for her neck. If reassurance alone proves insufficient, consider the possibility of physiotherapy, consisting of relaxation exercises, coupled with advice on the pursuit of daily activities.

Natural history and effect of treatment in cervicobrachialgia

As already mentioned, cervicobrachialgia occurs mainly between the age of 40 and 65 years, whereas spondylosis and spondylarthrosis occur progressively after the age of 40 years. Cervicobrachialgia therefore seems to be a symptom of *incipient* spondylosis and/or spondylarthrosis. It might even precede the first radiological signs. As the degenerative changes in the lower cervical spine progress, the pain disappears as a rule, whereas limitation of neck mobility progresses.

There is not the slightest evidence that drug treatment, radiotherapy, ultra-shortwave, massage, or any other treatment thus far available have any effect on the course of cervicobrachialgia. Forceful exercise treatment, traction, and manipulation might even prolong its painful course. In very refractory cases, immobilisation of the neck by means of a collar may be worth considering.

Case 2

Mr. P., a 32-year-old married clerk with one child:

"Doctor, my left arm aches and I have a tingling sensation in my left hand. It wakes me up at night."

Having said this, he lapses into silence with a worried expression on his face. So you ask:

"How long have you had this?"

"It started about a month ago."

"Had something out of the ordinary happened then?"

"It came on after I had been painting the ceiling."

When you then discover that Mr. P. is left-handed, you realize that this last answer may perhaps provide an important clue. The patient is too young for "ordinary" cervicobrachialgia, despite the fact that his complaints might appear to point in this direction. For a clerk, painting the ceiling implies prolonged confrontation with an unusual challenge! A neurovascular compression syndrome could therefore well be the correct diagnosis.

Neurovascular compression syndromes

Neurovascular compression syndromes of the upper extremities are due to entrapment of the main arterial and nerve trunks somewhere in the retroclavicular and axillary region. In most cases compression of the artery and the nerve occur only when the neck and/or arm are in a certain position.

Depending on the exact site of the compression, a distinction can be drawn between three main forms of neurovascular compression syndrome:

Syndrome	Site of compression
Scalenus syndrome	Between the scalenus anterior muscle and the first rib (occasionally a cervical rib)
Costoclavicular syndrome	Between the first rib and the clavicle
Hyperabduction syndrome	Between the pectoralis minor muscle and the ribs.

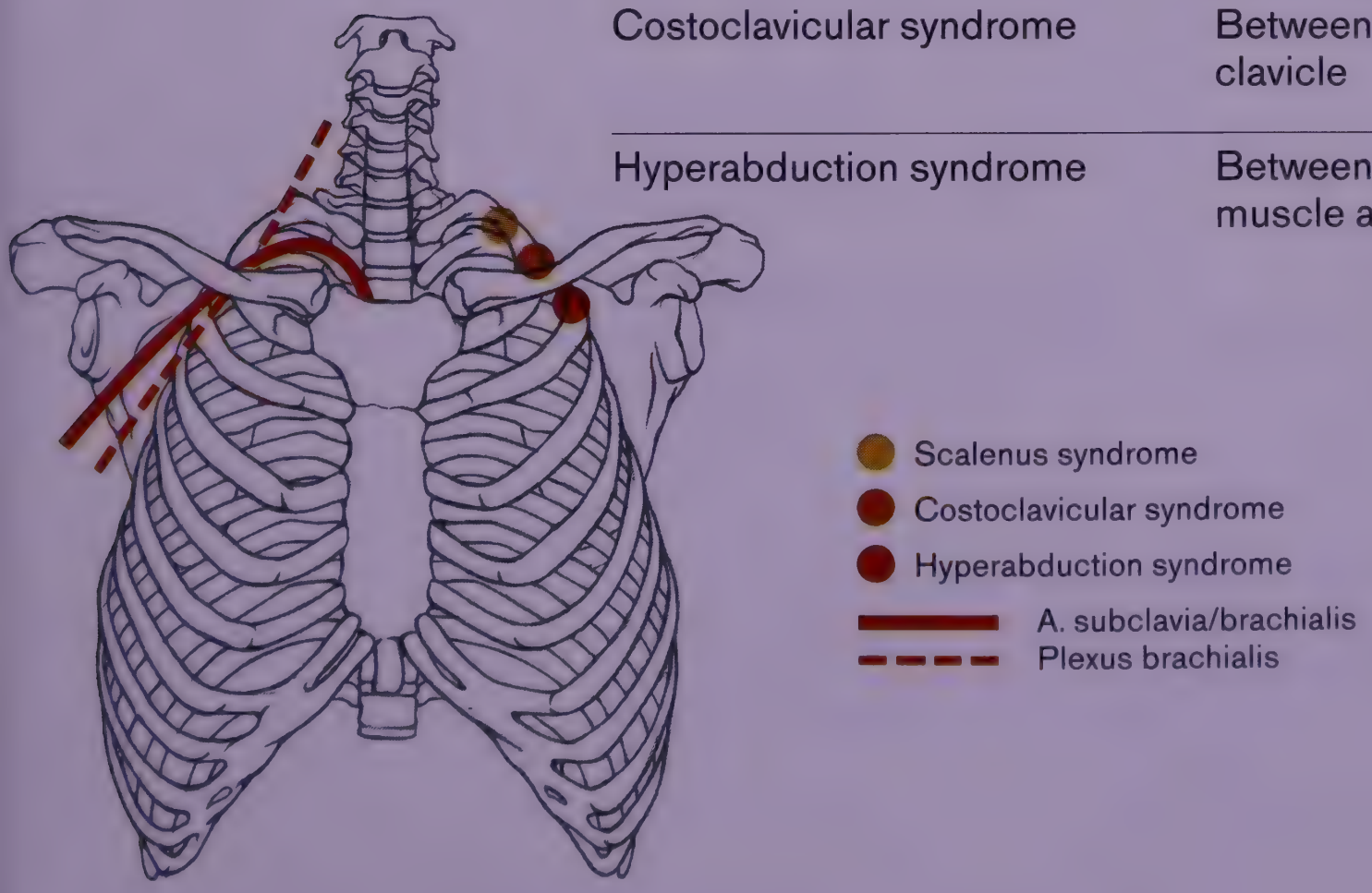


Fig. 6. Compression sites

Case history

Some additional questions may help you to find more evidence of a neurovascular compression syndrome and also to differentiate between the three varieties:

"When you wake up at night, do you notice that your arm is in one particular position?"

"I usually wake up with my hand behind my head, and the hand is numb."

"Have you ever noticed that your hand is white or cold?"

"It often feels very cold."

Physical examination

You now have strong grounds for supposing that the patient does indeed have a neurovascular compression syndrome, most probably of the hyperabduction variety.

Inspection

Look for swelling of the left hand.	None.
Look for discoloration of the left hand.	None.

Palpation

Are both hands equally warm?	Yes.
Are there symmetrical pulsations of the radial arteries?	Yes.

Auscultation

Listen for systolic murmurs in the supraclavicular area.	There are no murmurs.

Neurological examination

Test the arm reflexes and the sensibility of the upper extremities.	Normal.

As your standard physical examination has disclosed no abnormalities indicative of neurovascular compression, you should now proceed to the most crucial part of the examination, i.e. to the so-called provocation tests.

Provocation tests for neurovascular compression

The best way to establish the presence of a neurovascular compression syndrome is by eliciting signs and symptoms at relevant sites in the patient's neck and arm.

The signs and symptoms for the three forms of neurovascular compression in the upper extremity can be provoked as follows:

Costoclavicular syndrome

"Exaggerated military position": Back and neck stretched as much as possible and arms forcefully adducted to the body.

Hyperabduction syndrome

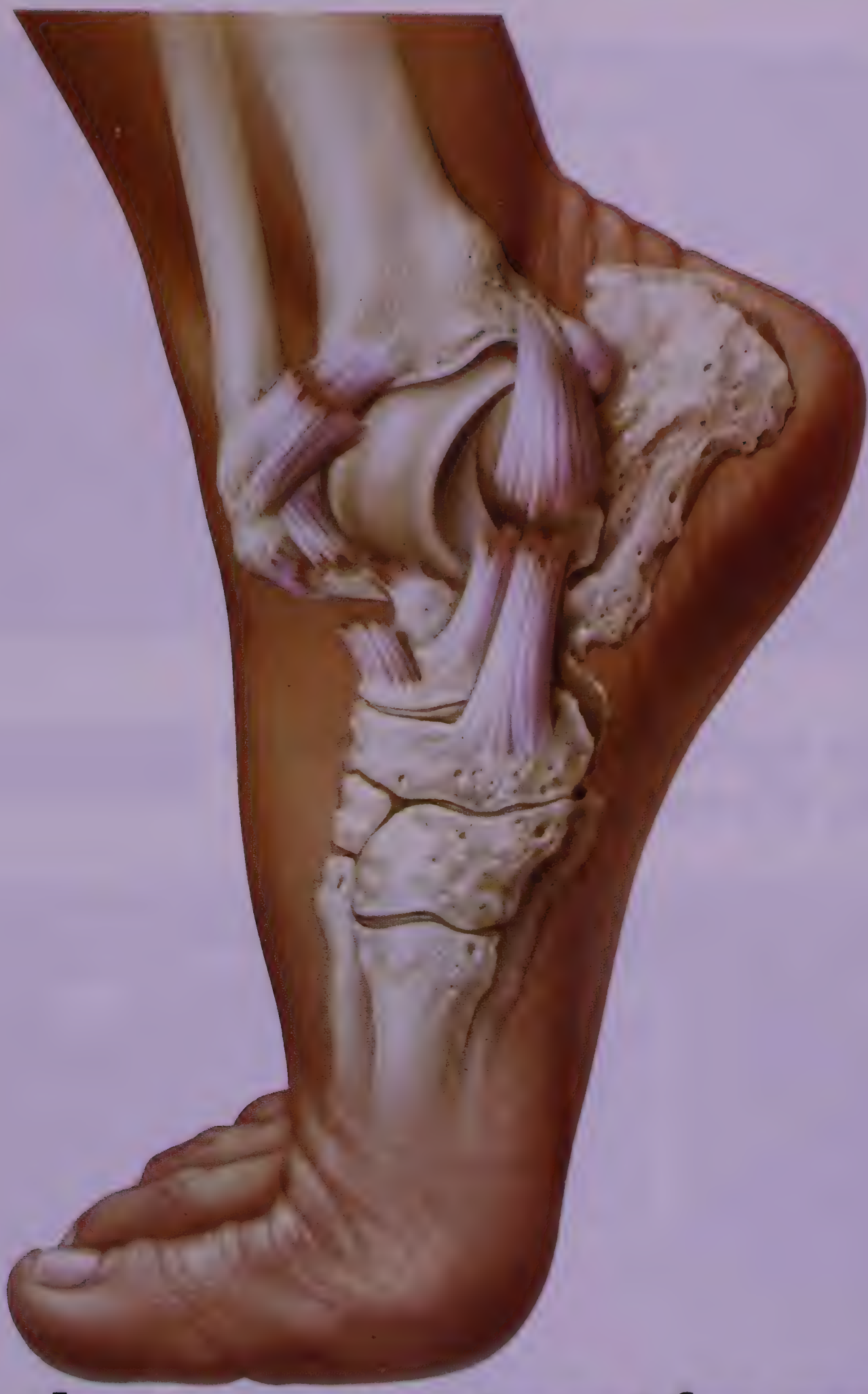
Patient sits with neck bent forward and arms hanging loosely by the sides. When the arms are raised as far as possible (abduction) the radial pulse fades or disappears and/or a systolic murmur becomes audible in the subclavicular area (Fig. 7).

Scalenus syndrome

Patient sits in relaxed position with arms hanging by the sides, head pushed back, and breath held after inspiration (Fig. 8). The chin is turned towards the symptomatic side (positive result indicated by fading or absence of radial pulse and return to normal pulse in normal position).

Fig. 7. Provocation test for the hyperabduction syndrome

Fig. 8. Adson test



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1. Van Heerden, J.J.: Diclophenac Sodium, Oxyphenbutazone and placebo in sports injuries of the knee. S.A. med J. 52 (10) p. 396-399, 1977.

Pointers in favour of the diagnosis

In your patient, neither the "exaggerated military position" nor the Adson test provokes any signs or symptoms, but during hyperabduction of the left arm he reports a marked worsening of his complaints. At the same time, you notice that the left radial artery pulse is no longer palpable; in addition, a loud systolic murmur now becomes audible when the stethoscope is placed in the left supraclavicular area.

Neurovascular compression

A diagnosis of one of the three neurovascular compression syndromes should only be regarded as confirmed if the following three phenomena occur during the provocation test in question:

1. Worsening of the patient's characteristic complaints: pain radiating through the arm and paraesthesiae in the hand.

2. Disappearance of the pulsation in the radial artery (and, eventually, bleaching of the hand).

3. Onset of a systolic murmur in the supraclavicular area.

Only when all three phenomena occur is a diagnosis of neurovascular compression warranted. Do not make the diagnosis simply on the basis of disappearance of the radial artery pulse!

Treatment

What treatment you should give will depend on how serious the signs and symptoms are:

1. If there are signs of ischaemia – even transient – in the arm and hand, a specialist (preferably a surgeon specialising in vascular surgery) should be consulted. He may possibly recommend arteriography of the arm and perhaps decide in favour of surgical treatment, although such treatment proves necessary only in a small percentage of patients.
2. In the great majority of cases, the complaints will disappear once the patient has learned to avoid placing the neck and/or arm in a position which triggers off his symptoms. Sometimes a short course of instruction by a physiotherapist may be helpful.
3. Particularly in cases of a clear-cut scalenus or hyperabduction syndrome, radiography of the upper thoracic aperture might reveal a cervical rib. If so, the possibility of its surgical removal should be discussed with a surgeon.

Case 3

Mrs. G., a 65-year-old widow, enters the consulting room with a pained expression on her face:

"For some days now I've had a terrible pain in my left shoulder. It's so bad that I can't move the arm. Now my hand is also beginning to hurt. And, look, Doctor, it's swollen too. Yesterday I only just managed to take my ring off. Today the fingers are even more swollen."

Mrs. G., whom you have known for a long time, has been suffering for the last few years from diabetes mellitus, for which dietary measures have proved sufficient as treatment. Having checked that she has not recently sustained a trauma, there is not much more you need ask her. The diffusely swollen hand and the apparently severe pain in the shoulder suggest a shoulder-hand syndrome.

Clinical picture of shoulder-hand syndrome

Shoulder-hand syndrome is a rather uncommon condition, presenting as a frozen shoulder combined with signs and symptoms of sympathetic dystrophy of the hand of the same side. The hallmarks of sympathetic dystrophy are:

- Hyperaemia
- Hyperalgesia
- Hyperhidrosis
- (Hypertrichosis)

It often sets in suddenly and is mostly seen in people over the age of 60 years. It seems to occur somewhat more frequently in patients suffering from diabetes mellitus or epilepsy, as well as in alcoholics.

Fig. 9. Normal external and internal rotation of the shoulder

Physical examination

Inspection and palpation

You start by looking at the patient's hand, the fingers and dorsum of which show marked, diffuse swelling. The fingers are held slightly apart and in a position of flexion. When you touch the hand, it is obvious that even this already causes pain. The hand is also warm and wet.

Mobility

Both passive and active movement of the fingers is impossible. You cannot determine whether this is simply due to the pain or whether there are genuine contractures.
After the patient has been helped to bare her left shoulder, you find that it is not swollen, red, or warm; but all passive movements are very painful. When you gently handle the arm, you note that passive abduction and anteflexion are restricted. The most conspicuous finding, however, is that external rotation is quite impossible, whereas internal rotation is normal and not painful.

These findings accord with your suspicion that the patient is suffering from shoulder-hand syndrome.

Diagnosis of shoulder-hand syndrome

The diagnosis of shoulder-hand syndrome is based mainly on the clinical picture, although radiography may be helpful in confirming the clinical diagnosis. An X-ray of the involved hand might show characteristic spotty decalcification of the bones (so-called Sudeck's atrophy), but it may take some weeks before this becomes apparent. Usually the X-ray of the shoulder reveals no abnormality.

Shoulder-hand syndrome is not associated with any rise in the erythrocyte sedimentation rate and there are no pathognomonic laboratory tests.

You now tell the patient that her condition is due to a temporary disturbance affecting her nervous system, which might have something to do with her diabetes. You warn her that the trouble may persist for quite a long time, but that it will eventually clear up. You add that there is not much that you - or any doctor - can do to arrest or to suppress the process.

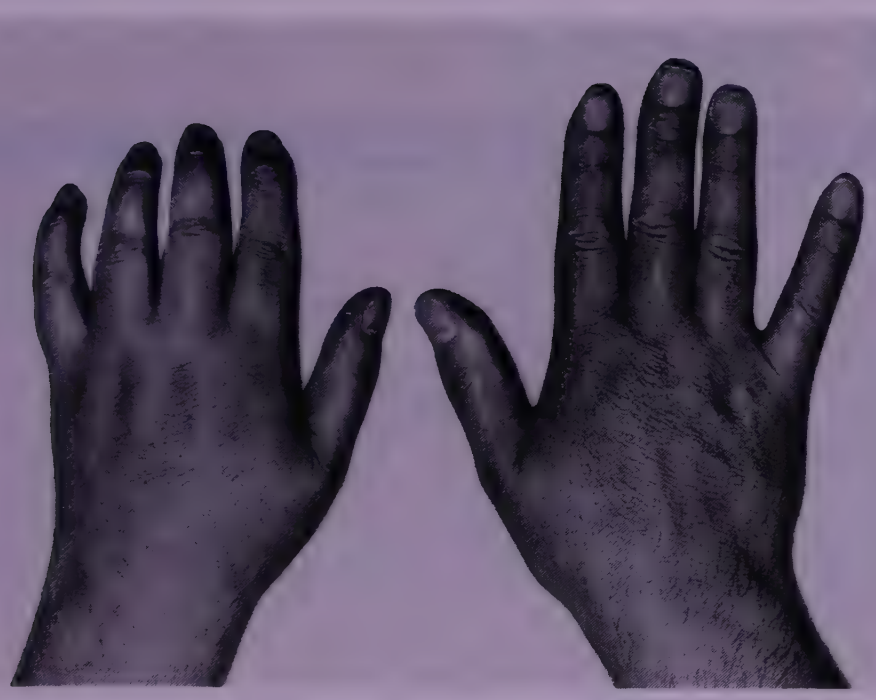

Fig.10. Sudeck's atrophy of the hand

Treatment for shoulder-hand syndrome

Although NSAIDs generally prove of little help as symptomatic treatment for shoulder-hand syndrome, they should be tried first before deciding to prescribe prednisone (10–20 mg daily). Prednisone seems to be the only drug which can be relied upon to afford relief in cases of sympathetic dystrophy.

Stellate ganglion blockade, sympathectomy, and other aggressive therapeutic procedures should be avoided, since there is no evidence that they have any favourable influence on the course of the process.

Exercise treatment can only be started provided the patient does not find it too painful; it should be gently intensified depending on the patient's powers of endurance.

Natural history of the shoulder-hand syndrome

As a rule two phases can be discerned in the course of this syndrome. On the average, both phases last - at a rough estimate - about six months: the first is a hypertrophic stage, in which there is much pain and swelling, and the second an atrophic stage, in which the pain and swelling subside and the hand becomes pale and cold. During the second stage, movement of the shoulder and fingers is still severely restricted, and it will improve only very slowly. In severe cases, complete restoration of mobility may take several years.

Although you can offer the patient no cure for her troublesome condition, you will try to alleviate the pain as far as possible, even though this will not be easy in the first few months. You will begin by prescribing a potent NSAID, to which you may possibly later add a low dose of prednisone.

You advise the patient to try at all times to keep her arm and hand in a comfortable position, e.g. by wearing a sling. You also tell her to move her fingers and shoulder a few times a day insofar as the pain permits. You explain to her that later, when the pain has subsided, you will arrange for a physiotherapist to give her exercise treatment.

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Folia rheumatologica

Rheumatology in everyday practice

Early stages of rheumatoid arthritis

M. Mahrenholtz
H. Zeidler

Vol.6

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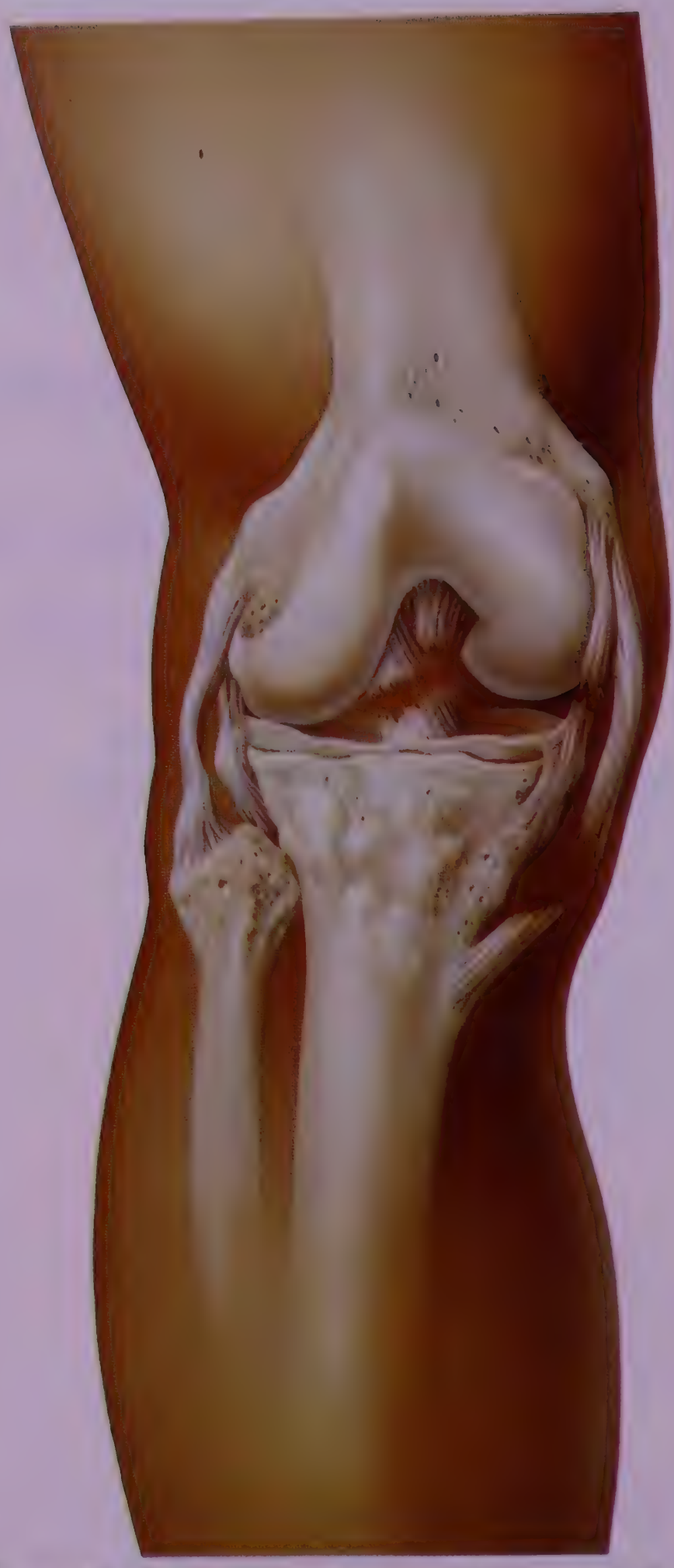

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Rheumatoid arthritis

Early stages, history of less than one year

Case studies

M. Mahrenholtz

H. Zeidler

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Case 1

Typical onset

Medical history

32-year-old patient, housewife with 3 children; last confinement 10 months ago; 4 weeks later for the first time painful swelling of the metacarpophalangeal and proximal interphalangeal joints, and pain in the metatarsophalangeal joints.

"It's worst in the morning; my fingers are so stiff that I can barely make the children's breakfast. It doesn't improve until late morning."

General, non-specific symptoms are also present, such as tiredness, reduced performance, and depressed mood.

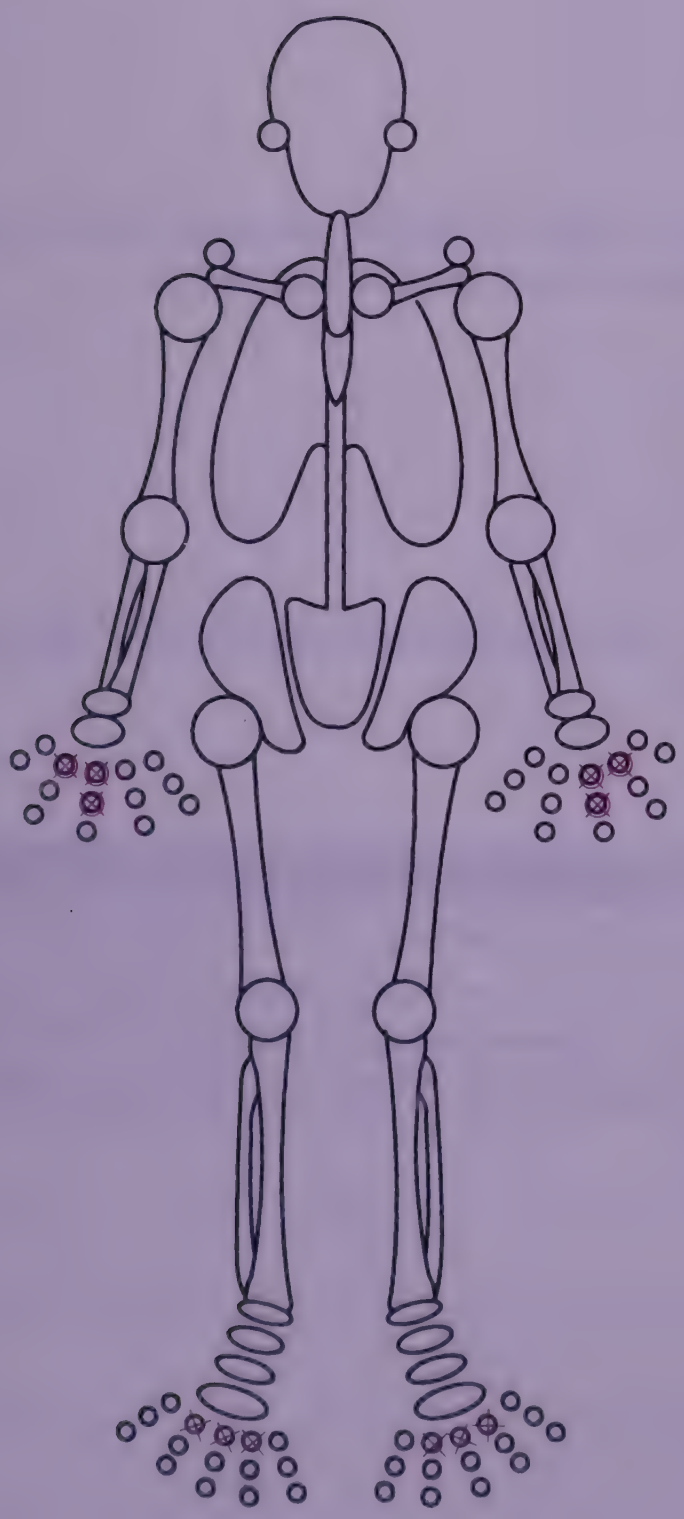
"What bothers me most is that I can hardly lift my baby because my hands are too weak."

The patient says that in cold weather she sometimes loses the sensation in her fingers.

Clinical examination

Discrete, symmetrical, tender swelling of the proximal 3rd interphalangeal joint and of the 2nd and 3rd metacarpophalangeal joints; pain in response to lateral pressure exerted on the metatarsophalangeal joints; tenderness of the 2nd, 3rd, and 4th metatarsophalangeal joints.

Fig.1. Swelling of the small joints of the hand



- ○ Swelling
- IIII Erythema
- ≡ Warm joints
- ~ Effusion
- ᴡ Crepitation
- △ Deformity
- × Tenderness and pain on percussion
- ≠ Limited range of movement
- ⊬ Pain on reaching maximum range of movement
- /// Myogelosis
-)(Atrophy

Differential diagnosis

Collagenosis (Raynaud's phenomenon, skin eruptions, signs of visceral involvement, antinuclear antibodies?)

Laboratory tests

Abnormal findings:

Rheumatoid factor positive in the latex agglutination test, 1:80 in the Rose-Waaler test
ESR 28/43 mm (Westergren method)
CRP 1.5 mg/dL

Normal findings:

Hb 14.7 mg/dL
WCB 6,200 × 10^6/L
Thrombocytes 180,000 × 10^6/L
Electrophoresis normal
ANA negative
Creatinine 75 µmol/L
GOT 6 U/L
GPT 8 U/L
GGT 12 U/L
Iron 15 µmol/L

X-rays

Hands and forefeet: no erosive changes.

ARA criteria, 1987 (Arnett et al)

1. Morning stiffness lasting at least 1 hour
2. Arthritis (observed by a doctor) in 3 or more joints in 14 possible sites (e.g. right or left PIP, MCP, wrist, elbow, knee, ankle, or MTP joints)
3. Arthritis in at least one site in the hands (wrist, MCP, or PIP joints)
4. Symmetrical arthritis, i.e. with simultaneous involvement of the same joints on both sides of the body (bilateral involvement of wrist, MCP, and PIP joints does not have to be absolutely symmetrical)
5. Rheumatoid nodules
6. Rheumatoid factors present in the serum
7. Radiological changes (erosion or osteoporosis around the joints)

At least 4 of the 7 criteria must be met for a diagnosis of rheumatoid arthritis. Criteria 1 to 4 must have been present for at least 6 weeks.

In the case of this woman criteria 1 to 5 are met, justifying a diagnosis of rheumatoid arthritis.

Treatment

- The doctor explains to the patient that rheumatoid arthritis is usually a chronic condition, progressing insidiously or in exacerbations, but that spontaneous remissions are also possible. He tells her that rigorous treatment in the early stages can check the progress of the disease, i.e. damage to the cartilage and, later, to the bone. He emphasises that the muscles and joints must be continually exercised in order to maintain mobility.
- Certain anti-inflammatory drugs may be taken by breast-feeding mothers. These include diclofenac*, flurbiprofen, and ibuprofen, because these substances have a short plasma half-life.
- Long-acting antirheumatics (i.e. "basic treatment") should not be taken by breast-feeding mothers or by any woman planning to become pregnant.
- Social services can arrange for a home help to come and look after the children on an hourly basis.
- The doctor prescribes 12 cryotherapy sessions followed by physiotherapy.
- Diclofenac (3 × 50 mg daily) is prescribed as a non-steroidal anti-inflammatory drug.
- The patient is advised to wean her baby. After weaning, chloroquine is prescribed as basic treatment; the initial dose is 2 × 250 mg daily for the first 14 days and the maintenance dose will be 1 × 250 mg. Every 4, and later every 8 weeks during treatment the patient is asked to give details of whatever side effects she has experienced. The blood count, liver enzymes, creatinine values, and kreatine kinase are also monitored. The patient should undergo an eye examination every 3 months.

* Following oral doses of 50 mg administered every 8 hours, the active substance passes into the breast milk, but in quantities so small that no undesirable effects on the infant are to be expected.

Use of antirheumatic drugs during pregnancy and lactation

Substances	Pregnancy	Lactation
Non-steroidal anti-inflammatory drugs	flurbiprofen ibuprofen ketoprofen	diclofenac* flurbiprofen ibuprofen
Corticosteroids	yes	yes
Antimalarial agents	no	no
Gold:		
parenteral	yes (with caution)	controversial
oral	no	no
D-penicillamine	no	controversial
Sulphasalazine	yes	yes
Cytostatics	no	no

From: Hartmann/Wittenborg/Zeidler, Praktische Rheumatologie

* Following oral doses of 50 mg administered every 8 hours, the active substance passes into the breast milk, but in quantities so small that no undesirable effects on the infant are to be expected.

Case 2

Atypical onset

Medical history

45-year-old patient; no serious diseases previously; painful swelling in the right knee for 6 months.

"Sometimes I can't sleep at night because of the pain."

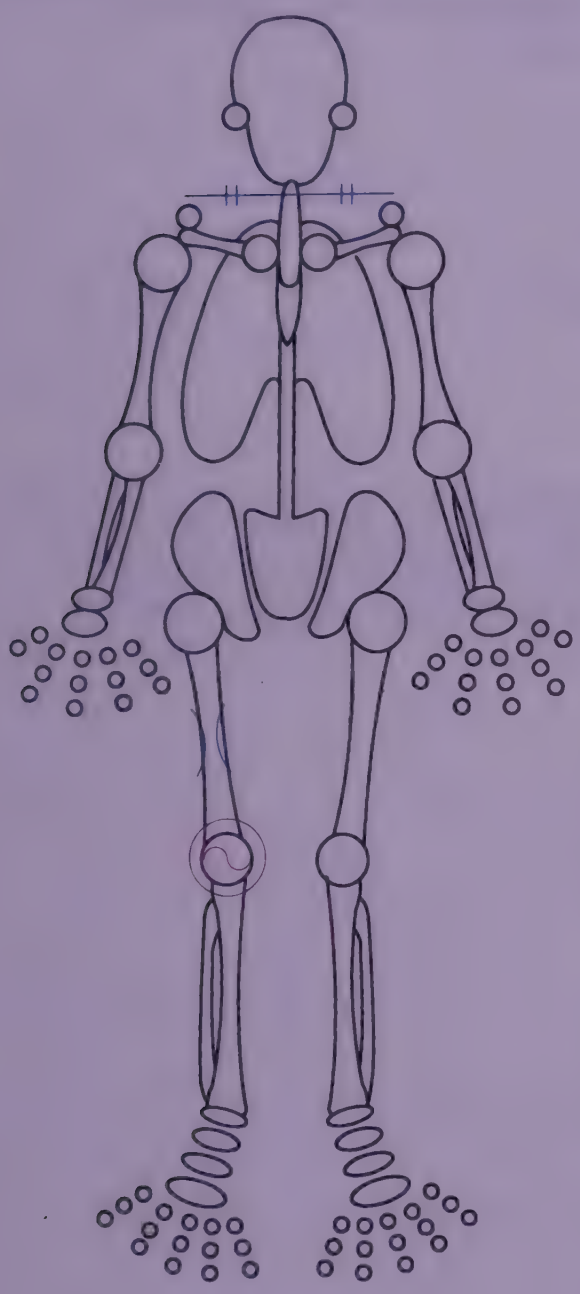
Pain in the neck on turning the head.

"I can hardly reverse my car."

Clenching the fists causes pain in the palms, and the metatarsophalangeal joints hurt on walking; the patient has had no backache, gastrointestinal symptoms, or problems with the urethra recently.

Clinical examination

Bilateral fluctuating swelling of the finger flexor tendons in the 4th tendon compartment, fluctuating swelling of the right knee joint, which is red and hot, inhibiting stretching and bending of the leg; taut elastic swelling of the popliteal fossa; overall range of motion of the cervical spine slightly limited.



Taut, elastic swelling of the popliteal fossa points to a Baker's cyst.

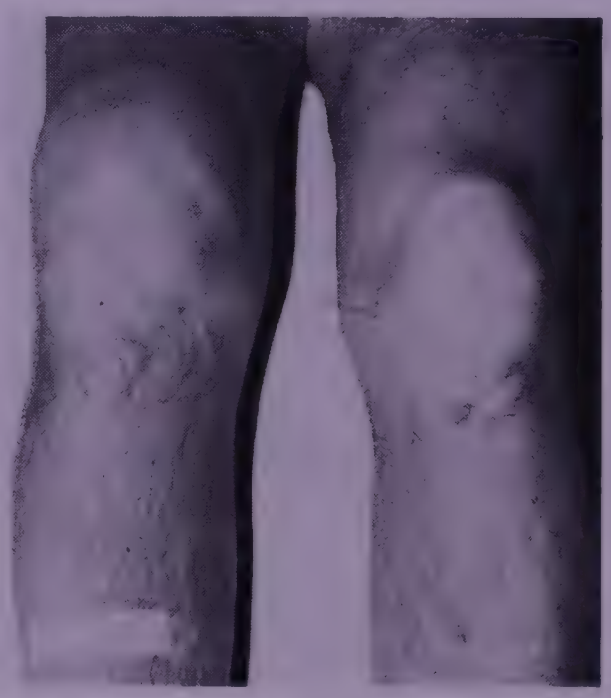
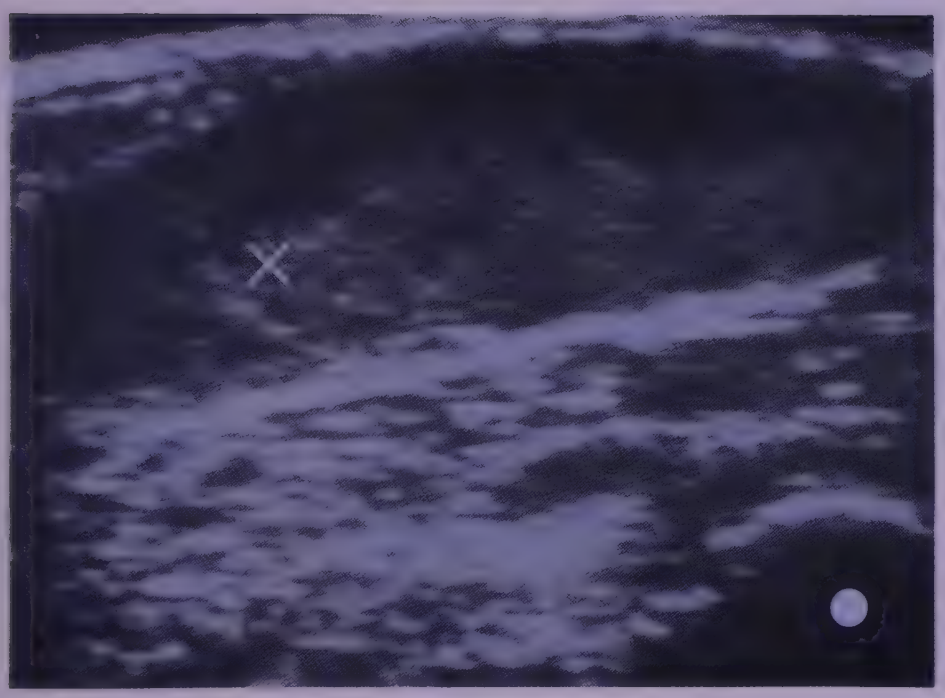


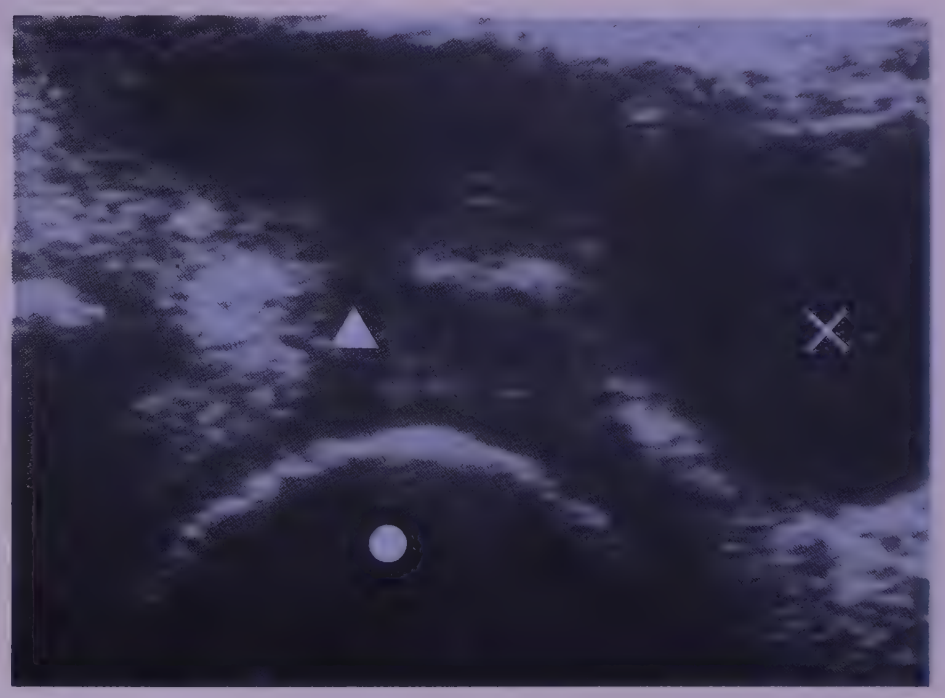
Fig. 2. Swelling of the knee joint

Fig. 3. Baker's cyst

Fig. 4. Ruptured Baker's cyst

Warning:
It is important to distinguish a Baker's cyst from thrombophlebitis. The typical signs of a ruptured Baker's cyst are knee joint effusion, followed by painful swelling of the calf, which may be red and warm. For this reason the knee joint should be examined by ultrasonography before phlebography is performed.





Figs. 5/6. Ultrasonographical view of a Baker's cyst = (×). Head of the femur = (●). Link between joint space and Baker's cyst = (▲).

Ultrasonography of the right knee joint

Large Baker's cyst in the subpopliteal recess, echo pattern partly weak and partly dense (indicative of a high fibrin content), no segmentation.

Differential diagnosis

- Seronegative spondylarthropathy (back pain, sacroiliitis on x-ray, family history, presence of HLA-B27 antigen?)
- Reactive arthritis/Reiter's syndrome (history of infection, eye disease, skin lesions, serology?)
- Psoriatic arthropathy (skin lesions, nail changes?)
- Sarcoidosis (pulmonary symptoms, thorax x-ray, skin lesions?)

Laboratory tests

Abnormal findings:

ESR 34/62 mm (Westergren method)
CRP 2.7 mg/dL
Gamma globulin 22%
Rheumatoid factors positive in the latex agglutination test, 1:256 in the Rose-Waaler test

Normal findings:

Hb 15.1 mg/dL
WBC 4,900 × 10^6/L
Thrombocytes 213,000 × 10^6/L
Creatinine 59 µmol/L
GOT 10 U/L
GPT 13 U/L
GGT 16 U/L
Iron 26 µmol/L
ANA negative
Antibodies against yersinia, chlamydia, and borrelia negative

Other findings:

HLA-B27 negative
Analysis of synovial fluid from the right knee joint: 6,800 cells/mL (72% granulocytes, 22% lymphocytes, 6% monocytes)

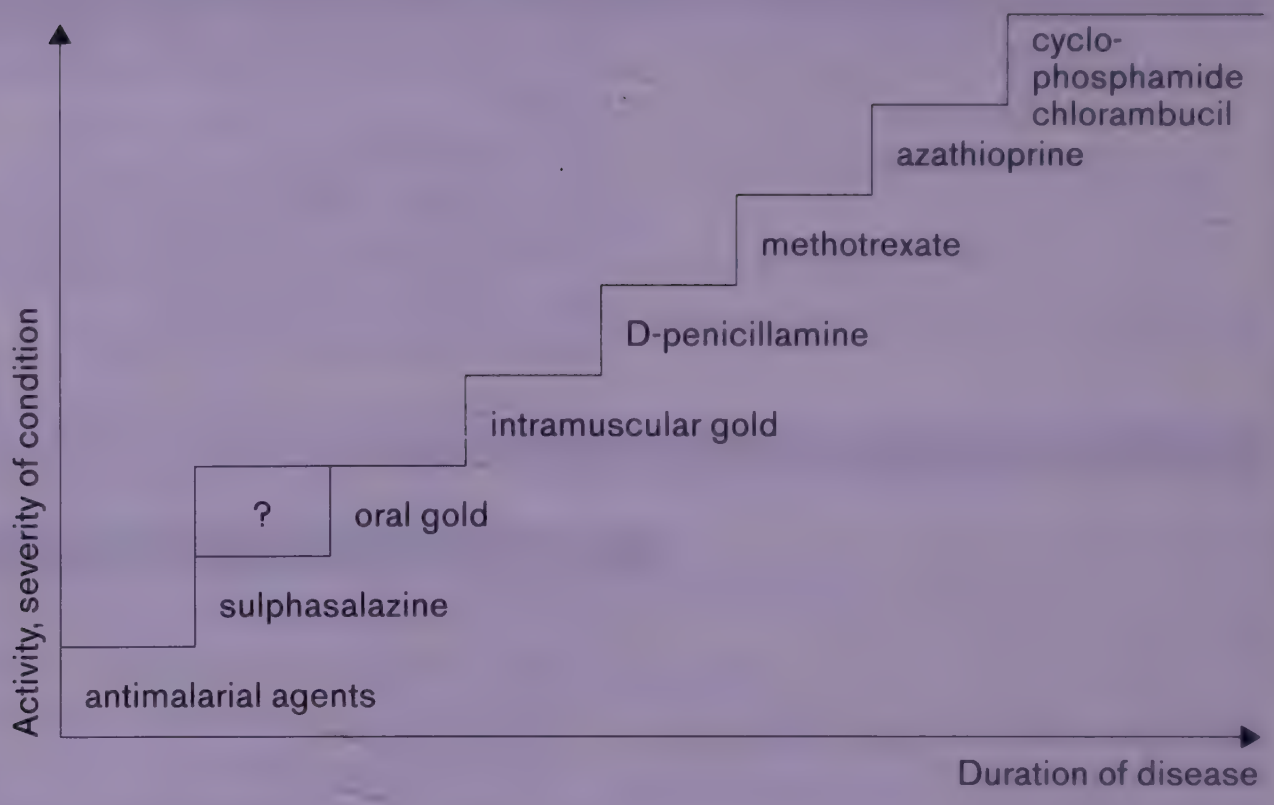
X-rays

Forefeet: incipient erosive changes in the metatarsophalangeal joints.
Hands: no erosion.
Knee joints: effusion on the right, otherwise normal.
Cervical spine: slight spondylotic bony spur, no erosive changes; functional x-rays normal.
Pelvis: sacroiliac joint spaces free, no sacroiliitis.
Lumbar spine: no inflammatory changes.

Treatment

- Chronic disease has already caused visible damage to cartilage and bone. In addition, mobility of the knee joint may be severely compromised. The patient is told that he is in urgent need of physiotherapy and drug treatment.
- A drug such as diclofenac (2 × 25 mg during the day and 100 mg at night) or indomethacin (same dosage) may be prescribed as a non-steroidal antirheumatic.
- As basic treatment the patient is given sulphasalazine (see graduated treatment plan) in gradually increasing doses, i.e. 500 mg daily in the 1st week, 2 × 500 mg daily in the 2nd week, 3 × 500 mg daily in the 3rd week, and a maintenance dose of 4 × 500 mg daily from the 4th week onwards. Every 4 weeks - and later every 4 to 6 months - the patient is asked to report any side effects he has experienced, and a complete blood count and urinalysis are carried out; liver enzymes GPT, ALP, and GGT are also monitored. The patient is told that the drug may cause reversible infertility.
- The doctor prescribes 12 physiotherapy sessions in which particular attention is to be paid to the right knee joint, including isometric exercises for the quadriceps muscles.
- If no reduction in the swelling is evident after 4 weeks, 40 mg of triamcinolone hexacetonide is injected into the right knee joint.

Graduated treatment plan for basic long-term treatment with antirheumatic agents (from Hartmann/Wittenborg/ Zeidler: Praktische Rheumatologie)



Case 3

Systemic onset

Medical history

63-year-old patient; for the past 4 weeks bilateral painful swelling and hotness of wrist and shoulder joints, metacarpophalangeal and proximal interphalangeal joints, and right ankle; weight loss of 6 kg during this time; dyspnoea and coughing; fever, sweating at night; general malaise.

Clinical examination

General state of health suboptimal, temperature 38.7 °C, symmetrical swelling of the wrist, metacarpophalangeal, proximal interphalangeal, and shoulder joints, as well as swelling in the right ankle; dullness above both lower lung fields, liver a handsbreadth below the costal arch, spleen palpable against the costal arch.

Differential diagnosis

- Vasculitis (acral necrosis?)
- Systemic lupus erythematosus
- Mixed connective tissue disease
- Acute viral infection (serology?)
- Paraneoplastic syndrome (careful history-taking; tumour markers, testing for faecal occult blood, etc.)

Laboratory investigations

Abnormal findings:

ESR 86/135 mm (Westergren method)
Hb 10.2 mg/dL
WBC 15,400 × 10^6/L
Thrombocytes 760,000 × 10^6/L
Iron 10 µmol/L
GOT 32 U/L
GPT 45 U/L
GGT 61 U/L
CRP 7.4 mg/dL
Alpha-1-globulin 6%
Alpha-2-globulin 9%
Gamma globulin 23%
Rheumatoid factor positive in the latex agglutination test, 1:1024 in the Rose-Waaler test
ANA positive (titre 1:60, speckled pattern)

Normal findings:

Creatinine 76 µg/L
Anti-DNS antibodies and crithidia reaction negative
Antibodies against chlamydia, yersinia, borrelia, rubella, measles, parvovirus, Epstein-Barr virus, hepatitis B cytomegalovirus, and HIV negative
Ferritin 350 µg/L
Transferrin 270 µg/dL

Analysis of synovial fluid from the right shoulder joint

WBC 23,600/mL (82% granulocytes, 14% lymphocytes, 4% monocytes), rheumatoid factors positive in the latex agglutination test and 1:1024 in the Rose-Waaler test, ANA positive, speckled pattern, titre 1:60, anti-DNS antibodies and crithidia reaction negative.

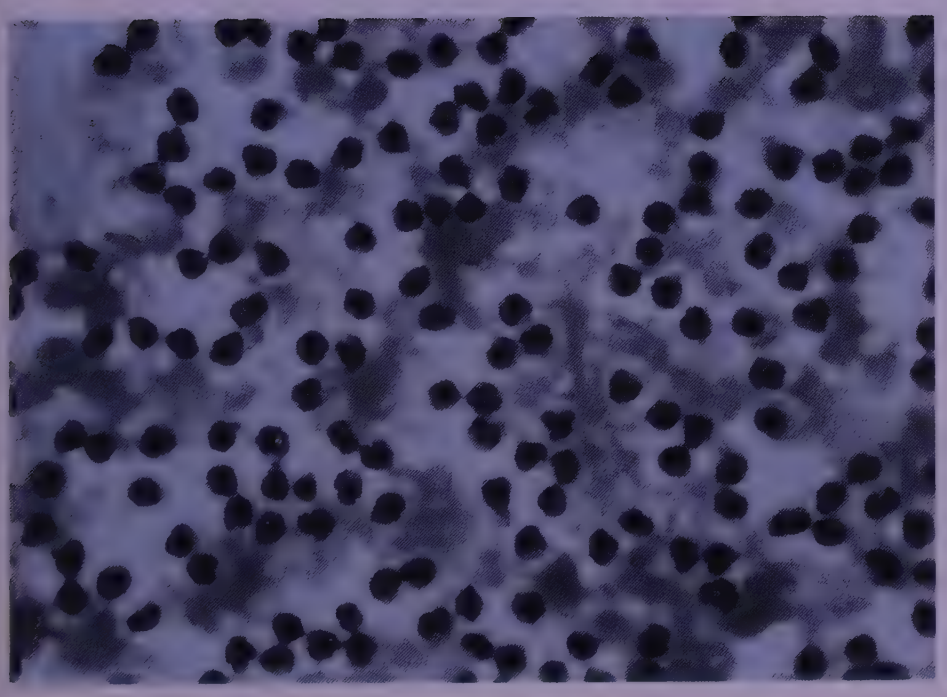
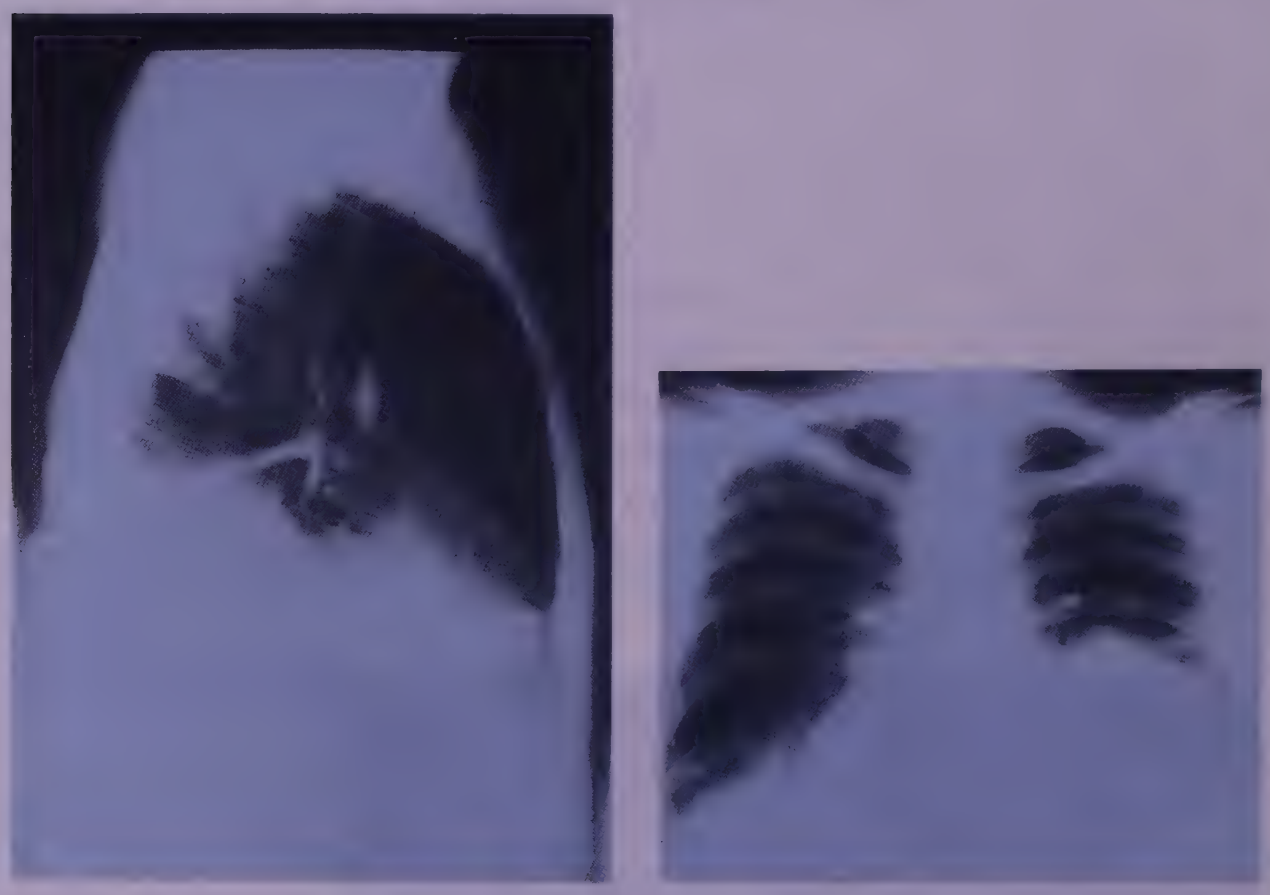


Fig. 7. Synovial fluid smear in inflammatory joint disease

X-rays of the joints

Hands, forefeet, shoulders, ankles, and cervical spine: no erosive changes.

X-ray of the thorax: bilateral pleural effusion.



Figs. 8/9. Bilateral pleural effusion

ECG: amplitude of QRS complex variable, low voltage.

Echocardiogram: pericardial effusion (Fig.10).

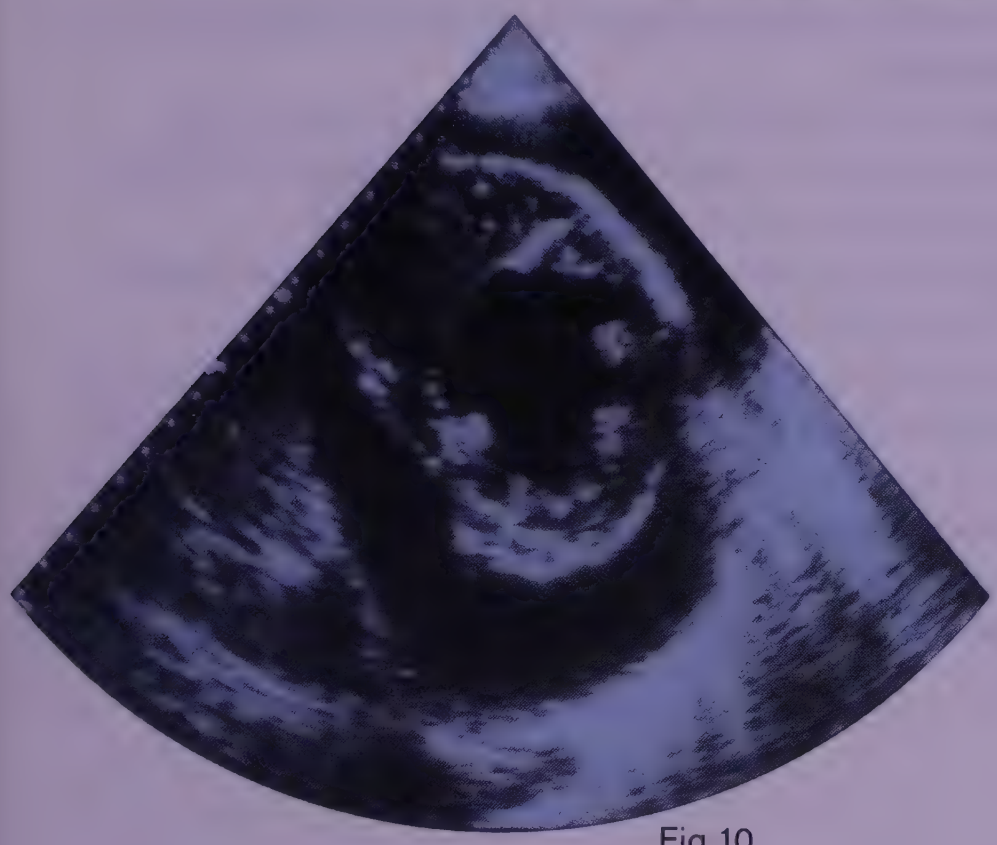


Fig.10

Lung function: vital capacity 3,500 mL, FEV_1 = 73% of vital capacity, i.e. not indicative of obstructive or restrictive airways disease.

Ultrasonography of the abdomen: homogeneous densification of liver and spleen, liver a handsbreadth below the costal arch, spleen enlarged ($6 \times 9 \times 12$ cm^3).

Treatment

- The patient is hospitalised immediately.
- Corticosteroid treatment is initiated (1 mg prednisone per kg body weight).
- In addition, an ordinary analgesic such as paracetamol can be given if necessary.
- Intramuscular injections of aurothioglucose are started (problem of patient compliance; aggressiveness of the disease and consequent risk of early joint erosion).
- Cryotherapy followed by intensive physiotherapy is already prescribed during hospitalisation.
- After discharge from hospital the patient's condition must be closely monitored at a rheumatology clinic. The dose of the corticosteroid can be gradually reduced in accordance with clinical and laboratory findings. Once a dose of 10 mg prednisone daily has been reached, the patient can be given 3×50 mg of the non-steroidal antirheumatic drug acemetacin in addition.

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1. Skoutakis, V.A.; Carter, C.A.; Mickle, T.R.; Smith, V.H. et al.; Review of diclofenac and evaluation of its place in therapy as a nonsteroidal antiinflammatory agent. Durg Intell and Clinical Pharmacy. 22:850-859, 1988.